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ORIGINAL LECTURES.

THE MIDDLETON GOLDSMITH LECTURES.

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LECTURE I.

MULTIPLE NEURITIS AND ITS RELATION TO CERTAIN PERIPHERAL NEUROSES.

IN inaugurating the course of lectures which bears the name of the founder, it is becoming that some expression of gratitude should be made to Dr. Middleton Goldsmith. This can hardly be associated with any form of personal encomium, since that would be as distasteful to him as it is needless before this Society, which owes its existence in part to his exertion. It is proper, however, to emphasize our appreciation of the motives which have led to the establishment of this lectureship. Every one knows that in the rapid progress of medical science, in the great and needful division of labor which that progress has made imperative, there is offered to each individual worker a vast mass of facts which cannot be easily grasped or even readily comprehended. To classify these facts, to weigh their importance, to draw logical conclusions from them, and to present these conclusions in a clear and accessible form is a work of no little magnitude, but of absolute necessity. To accomplish this specialists are at work in every department, and it may be well to emphasize the fact that it is the necessity, for their labor, rather than any temporary fashion, that has led to that specialization in medical science which is so often harshly criticised. But special studies lose half their value if they are not made generally available. And it is just here that a lectureship such as this has its use. It enables the results of individual labor to reach beyond the specialist, to be placed before the general practitioner in a simple but comprehensive way. There is, therefore, no means by which an individual can make his influence more widely and permanently felt than by the endowment of such a foundation. For when his personal activity has of necessity ceased with the natural termination of an honorable career he may be sure that, yearly, facts and results are proclaimed in his name of immeasurable service to his successors and of benefit to the profession.

And now, before proceeding to the subject which is to occupy our attention for a time, let me express my obligation for the honor which this Society has conferred in inviting me to inaugurate this course of lectures. It is an honor to which I have no claim, and the duty incurred is one which I feel incapable of performing satisfactorily. I must beg your indulgence in the performance of the task you have laid upon me, and if I succeed in eliciting your interest I know that it will be because the subject we are to study together has a novelty which may compensate for the defects of its presentation.

The discovery of a new disease is never made suddenly. It is a gradual process, and certain stages in the progress toward its complete recognition may be observed. There is first the period of clinical observation, when isolated cases of an unfamiliar and mysterious affection are recorded as curiosities. To this succeeds the period of diagnosis, when, by a comparison of the now numerous cases, a clinical picture of the disease is gradually filled out. In this stage there is much to impede the progress of discovery; for, not content with an analysis of symptoms, and a grouping of cases, the majority of observers offer theoretical explanations of the nature of the new affection, and an element of speculation enters which often obscures the facts. There is, however, a real progress in this period, for it is characterized by inductive reasoning from fixed data, and as a result reliable conclusions are reached which make a diagnosis possible. The third period is that of pathological discovery, where the morbid changes lying at the base of the disease are accurately ascertained. In this stage erroneous theories are eliminated, true explanations for various symptoms become self-evident, and the exact nature of the affection is determined. The disease has now a status of its own. And at first this might seem to be the final stage in the progress of discovery. But it is not. There remains a period of etiological classification, when conditions, formerly supposed to be dissimilar, are found to have a common basis, when the pathological changes are ascertained to be the same, although the clinical pictures have varied, and when classification of the various forms is rendered possible and a definition of the disease is reached.

Such a gradual advance toward general recognition is well illustrated by the history of multiple neuritis, which is to engage our attention at the present time.

I think we may claim that one of the first cases presented was by an American physician, Dr. James Jackson, of Boston, in 1822.[1] In a paper "On a Peculiar Disease Resulting from the Use of Ardent Spirits," which he named arthrodynia, he gives a most graphic picture of what we now know to be one form of multiple neuritis. He says, "This disease comes on gradually. It commences with pain in the lower limbs, but especially in the feet, and afterward extends to the hands and arms. The hands may be affected first in some instances, and in all cases in an advanced state the pain is more severe in the feet and hands than in the upper part of the limbs. The pain is excruciating, but varies in degrees at different times. It is accompanied by a distressing feeling of numbness. After the disease has continued a short time, there take place some contractions of the fingers and toes, and inability to use these

[1] New England Journal of Medicine and Surgery, vol. xi. p. 351.

parts freely. At length the hands and feet become nearly useless, the flexor muscles manifesting, as in other diseases, greater power than the extensors. The whole body diminishes in size, unless it be the abdomen, but the face does not exhibit the appearance of emaciation common to many visceral diseases. The diminution is especially observable in the feet and hands; and at some time the skin of these parts acquires a peculiar appearance. The same appearance is noticed in a slighter degree in the skin of other parts. This appearance consists in a great smoothness and shining, with a sort of fineness of the skin. The integuments look as if tight and stretched, without rugæ or wrinkles, somewhat as when the subjacent parts are swollen; but the skin is not discolored. Yet in this disease there is not any effusion under the skin, and the character which this assumes arises from some change in the organ itself. The most characteristic symptoms are manifested in the limbs, but the pain is not limited to these —and other symptoms are exhibited in other parts. The pain sometimes shoots suddenly up one or both legs, and in one case it frequently passed up the back and then forward to the pit of the stomach. The functions of the stomach are always impaired; the mind is weakened; sleep is prevented by pain. I believe that this disease is always fatal when the use of spirituous liquors is not abandoned before the powers of the digestive organs are greatly impaired."

It is hardly possible, even at the present day, to add to this description, which portrays in strikingly vivid language the main features of one form of multiple neuritis.

The next observations of importance in establishing a clinical picture were made by Magnus Huss, who, in his work upon chronic alcoholism, in 1852, gave a very complete description of alcoholic nervous symptoms, dividing the cases into paralytic, anæsthetic, convulsive, epileptic, and hyperæsthetic forms. But not content with the clinical side, he advanced to pathological hypothesis, and ascribed all the various symptoms to lesions in the central nervous system. His description was amplified and completed by Lancereaux, in 1864, in an article upon alcoholism in the *Dictionnaire Encyclopédique des Sciences Médicales*. But the pathology of the disease was still a matter of speculation.

In 1855 the great work of Duchenne was published,[1] and in it a number of cases are recorded which we now recognize as multiple neuritis. In these cases there were sensory disturbances, consisting of pain, numbness, and loss of sensation; motor disturbances, consisting of paralysis, with atrophy, especially marked in the distal parts of the extremities, and attended by a loss of faradic contractility in the paralyzed muscles; and cyanosis, coolness, and increased sweating in the affected limbs. Duchenne grouped these cases together under the title "Paralysie Générale Spinale Subaigüe Ascendante," because he thought a gradual advancing lesion in the spinal cord, from below upward, would explain the symptom. It is true that he found no macroscopic change in the cord in the only case in which he made an autopsy. But when, under the leadership of the French school, from 1860 to 1865, the microscopic appearances in nervous lesions began to be studied, the hypothesis of Duchenne at once appeared to be verified, for it was found that such symptoms as numbness, pain, and anæsthesia were associated with lesions of the posterior columns of the spinal cord. And it was also proven that atrophic paralysis was caused by a degeneration of the ganglion-cells of the anterior gray horns of the cord, not necessarily visible to the naked eye. It seemed an easy step to the conclusion that when these symptoms occurred together, the entire spinal cord was the seat of disease, and that wherever they occurred a spinal lesion was progressing. The pathology of this form of paralysis appeared to be definitely ascertained, and for many years the fallacy of such a conclusion was not detected. All atrophic paralysis was invariably referred to spinal lesions, because spinal lesions may cause atrophic paralysis.

But facts rarely accommodate themselves permanently to theories, and after a time a mass of very unwieldy facts began to accumulate. Cases of atrophic paralysis without spinal lesion were observed, and these threw doubt upon the theoretical pathology. The period of true pathological observation had begun, and gradually went on to completion. In 1864 Duménil reported the following case, which deserves to be cited, as it is the first in which an autopsy established the existence of a widespread disease in the peripheral nerves as a cause of sensory, motor, and trophic symptoms.

Observation I.—A tailor, aged sixty-one, after suffering from prickling in the toes for two weeks, was suddenly taken with weakness in the left arm and right leg, and a few days later by the same paresis in the left leg. Within five days he could not stand or walk. The paralyzed feet hung flaccid, and were totally paralyzed; the thighs could be moved freely. Anæsthesia was found on the right sole and calf, and on the left foot and outer side of the leg. In the muscles of the hands and forearms a considerable atrophy, with paralysis, developed. The faradic contractility was abolished in the paralyzed muscles. He complained of a painful numbness in the paralyzed limbs up to the knees, and involving the hands. No improvement; death in four and a half months.

Autopsy: Spinal cord and nerve-roots were normal. The finer nerve branches in the legs and hands were degenerated, only a small number of nerve-fibres being found. Single fibres showed no continuous myelin sheath, but this was segmented and granular. There was an increase of connective tissue, and many fat-cells in the nerves.[1]

This case being of an anomalous character excited very little attention. Two years later, however, Duménil reported another, and published an elaborate article on peripheral paralysis, in which he said: "My own observations have convinced me firmly that many paralyses of obscure origin are caused by true spontaneous neuritis."[2] The second case, which, with the first, led to this conclusion, has been cited by Leyden as a case of neuritis, but it does not conform to any of the types of multiple neuritis, and is open to some doubt from the results of the autopsy, since numerous foci of

[1] Electrization localisée.

[1] Duménil: Gaz. Heb., 1864, p. 203, and Gaz. Heb., 1866, No. 4.

[2] Ce que j'ai observé me donne la conviction intime que bien des paralysies de cause obscure ont leur point de départ dans les véritables névrites spontannées. Gaz. Heb., 1866, No. 4.

disease were found in the spinal cord. We, therefore, pass it by.

Singular as it may seem, an interval of ten years elapsed before another case of similar nature, accompanied by a record of *post-mortem* examination, appeared. Then Eichhorst, of Berlin, reported the following interesting history:

Observation II.—A female, aged sixty-six, after suffering daily for two weeks from a chill, fever, and sweat, attended by malaise, anorexia, and constipation, noticed an œdematous swelling of both feet and legs, and complained of pain in the abdomen. A week later, on admission to the hospital, these symptoms continued. The urinary examination was negative. Three days after admission she suddenly felt a severe boring pain in the left leg, shooting into the toes, and at the same time a profuse sweat broke out over the calf and back of the foot. A few hours later a total paralysis was found in the muscles supplied by the peroneal nerve, with a marked anæsthesia. The electric reaction, at first preserved, was found two days later to be gone. After six days, during which she had no further chills, a paralysis of the anterior tibial nerve developed, and soon after of the posterior tibial nerve also. One week later entire paraplegia of the legs, with anæsthesia, severe pains, continual sweating, increasing œdema, and loss of the tendon reflexes had ensued. There followed a paralysis of the left, and soon after of the right, radial nerve. Moderate fever continued, and albuminuria appeared. Three weeks after her admission sudden blindness developed, the ophthalmoscopic appearances being at the time normal; the patient then lay in bed with eyes closed, unable to move a limb. The extremities perspired constantly, and were tender, any pressure on nerve-trunks being very painful. In the face there was no trouble, and the senses were normal except that of sight. No trouble in swallowing. Nothing abnormal about the viscera during the entire disease. No irregularity of pulse or respiration. Death occurred on the forty-fourth day of the disease.

Autopsy: Spinal cord absolutely normal. The nerve-trunks in the bicipital grooves appeared intensely red to the eye, the perineurium being discolored and the endoneurium blood-red. The same appearance was noticed in the large nerve-trunks of the arm in their course, as well as in the left tibial nerve. The microscopic examination showed a remarkable distention and tortuosity of the bloodvessels of the perineurium; the vessel-walls were thickened, their nuclei increased. In the vicinity of the vessels a large number of lymphoid cells were found, which everywhere followed the vessels and infiltrated the connective tissue. There were also numerous fatty cells. The connective-tissue fibrillæ of the perineurium were thickened, shining, and swollen; their nuclei were increased and partly infiltrated with fat granules. Similar changes were seen in the endoneurium, viz., numerous extravasations of blood, which separated the nerve-fibres and compressed them. The nerve-fibres showed marked degeneration, especially those lying next the endoneurium, consisting of disintegration of the myelin sheath, and a distention and spindle-shaped swelling of the individual nerve-fibres. The nuclei of the sheath of Schwann were not increased in number, but the protoplasm about them was coarsely granular and opaque. The cells of the endoneurium were everywhere wanting, being replaced by fatty granular cells, even between the uninjured fibres.[1]

This is the first case to be found in which the microscopic appearances are described with sufficient detail to be satisfactory. It is to be noticed that here the lesion was an acute inflammation, and was, apparently, a diffuse one, both interstitial tissue and nerve-fibrils being involved in the process. That it was primarily an interstitial inflammation, and that the affection of the nerve-fibrils was secondary, due to pressure of the products of exudation, is clearly seen in the fact that those fibres were more seriously affected which lay near vessels, while the deeper fibres in large bundles were not at all degenerated. The appearance of the fibres was such as occurs in any degeneration from pressure. There is no reason, therefore, to believe that the process began diffusely.

As to the symptoms in this case, it is to be remarked that they were ushered in by an acute febrile movement with chills, and that severe pain was an early and prominent symptom; also, that œdema and sweating were present; and that the optic nerves were involved.

The clinical picture in the following case of Joffroy, published three years later, was somewhat different, as was also the pathological condition.

Observation III.—A washerwomen, aged thirty-three, in the last stage of phthisis, was admitted to the hospital on March 5th. In February she had noticed a rapidly increasing weakness in her legs, and at the time of admission she could not walk or raise her feet from the bed. She could flex the knees but not extend them. There was no contracture, and the muscles were relaxed and flabby. Sensations to pain, temperature, and pressure impressions, were normal, but the muscular sense was lost, and reflexes were diminished. She showed such a degree of mental weakness that tactile sense could not be tested. She had no shooting pains, no loss of control over bladder and rectum, no bedsores. Two weeks later the arms became involved in the paralysis, and atrophy, incoördination, and loss of muscular sense, with fibrillary motions, developed within a few days. There was great diminution of faradic excitability in all the paralyzed muscles. At the end of ten days the arms were entirely powerless, but retained their sensibility. On April 7th she died.

Autopsy: A chronic meningitis of the brain explained the mental symptoms. The spinal cord was normal. The nerves appeared normal; but microscopic examination showed very marked degeneration in all the nerve-trunks, but especially in the sciatic, radial, and ulnar nerves. There was a segmentation of the myelin sheath, which at places was reduced to a finely granular mass. Many sheaths of Schwann were filled with this mass, others were empty. The nuclei of the sheaths of Schwann were increased in number; all the spinal nerve-roots were normal. The changes in the nerves were followed down into the fine terminal branches in the thenar muscles. The muscles were atrophied and showed fatty degeneration.[2]

Here, in contrast with the preceding case, it is to be noted that the lesion was not attended by congestion of the nerves, or by any exudation of lymphoid cells, or by

[1] Eichhorst: Virchow's Archiv, Bd. 69, S. 265, 1876.

[2] A. Joffroy: Arch. de Phys. norm. et Path., 1879, pp. 172–198.

marked interstitial changes. The affection was a true parenchymatous inflammation, with degeneration of the myelin sheath and axis-cylinder. As a result, the macroscopic appearance of the nerves was not such as to attract attention, and it required a microscopic examination to demonstrate the changes present. Joffroy, who reports this case as one of general spontaneous neuritis, finds the lesion identical with that observed in cases of localized neuritis occurring from cold, from lead palsy, or as a sequel of the infectious diseases. In regard to the symptoms, also, the case contrasts strongly with the preceding one. The patient had phthisis. The disease advanced more slowly. Pain was absent, and the sensory symptoms were by no means prominent, the muscular sense being the only one affected.

In 1880, the following cases were observed by Leyden in the Charité Hospital of Berlin, in both of which changes were found in the peripheral nerves. They are cited because they not only enlarge our clinical picture, but confirm the pathological conditions already described.

Observation IV.—A sailor, aged twenty-five, was suddenly seized with severe tearing pains in all four extremities, especially near elbows and knees, which were swollen, white, indurated, and very tender. The pains shot down the limbs, and were attended by formication, and by diminished sensation in fingers and toes. High fever accompanied the onset. In a few days, as the fever subsided, a paretic condition with muscular atrophy was noticed in the forearms, and to a less extent in legs and feet. A gradual improvement took place in the course of a few months in the lower extremities, but the paralysis and atrophy in the arms increased in degree, and the appearance of the hands resembled that seen in lead palsy. There was a reaction of degeneration in the muscles paralyzed. A year after the onset he died of chronic nephritis.

Autopsy: A sclerotic atrophy of both radial nerves was found. The nerve-sheath was thickened. The muscles were considerably atrophied. The anterior spinal nerve-roots, as well as the cells in the anterior horns, and the entire spinal cord, were normal. In the nerves of the lower extremities no changes were visible.[1]

Observation V.—The second case was that of a merchant, aged thirty-one, who noticed about Christmas, 1878, a feeling of formication in the toes and sole, of first the left, and then the right foot. This continued more or less severe, without any motor disturbance, until July 10th, when he found his legs heavy, and in two days this had so increased that he could not walk. At the same time a constant formication in the soles of the feet was felt. Within a few days similar symptoms appeared in the arms and hands. On admission to the hospital on July 17th, he complained of weakness of the legs and loss of sensation in the hands, also burning pain in the left hand and inner side of the left knee. The motion of the legs was powerless and uncertain, and there was paresis of the extensors of the hands and fingers. Sensation was much impaired in the periphery of the extremities; the tendon reflexes were abolished, the muscles flabby. The electrical examination showed a loss of faradic contractility, and a great diminution of galvanic contractility in the affected muscles. The liver was tender, and there was a slight jaundice; pulse 70; no fever. The paralysis increased slowly in extent and severity, and was accompanied by paræsthesia, pains, and diminished sensibility. The muscles atrophied, and showed the reaction of degeneration. After six weeks slight fever began, with rapid pulse and dyspnœa. The pains affected the upper part of the thorax, and the spinal column became sensitive as low as the eighth dorsal vertebra. During the last days small rapid pulse, severe dyspnœa, purely costal respiration, delirium, somnolence, and exhaustion were noticed, and on September 3d he died.

Autopsy: The spinal cord was normal, the gray and white substance intact, the large ganglion-cells in normal form and number, though a few seemed to present a slightly swollen, glassy appearance which Leyden considered the first indications of trophic disturbance. The nerve-trunks, especially the radials and peronei, presented the appearance of a high degree of fatty degeneration and atrophy. The myelin sheath was either wholly wanting or greatly divided and split up, and in a state of fatty degeneration; the axis-cylinder seemed varicose from deposits of fat in it. A marked infiltration of lymphoid cells, especially between the endoneurium and nerve-fibres, was noticed, more marked along the course of the small arterioles. These changes were present in a greater degree in the peripheral branches of the nerves, and seemed less in the nerves near the cord.[1]

It will be noticed that here the lesion corresponded quite closely to that in the case of Eichhorst.

Impressed by the striking features of these two cases, and by their similarity, both in clinical symptoms and pathological changes, to the cases of Duménil, Joffroy, and Eichhorst already cited, Leyden undertook a review of atrophic paralysis.[2] He showed the confusion which had come from blindly referring all such cases to a single lesion in the spinal cord. He proved that previous authors had erroneously grouped together several diseases distinct from one another, both in their course and in their pathology. One of these is poliomyelitis anterior, acute and chronic, occurring in children and adults; a disease of the anterior gray horns, with local foci of inflammatory degeneration; a disease characterized by motor and trophic symptoms of peculiar distribution and typical course. A second of these is progressive muscular atrophy, with its corresponding bulbar affection, glosso-labio-laryngeal paralysis; a disease of the anterior gray horns and of the motor cranial nerve-nuclei, of a chronic degenerative, non-inflammatory kind; a disease characterized by trophic and motor symptoms of different distribution, nature, and progress from the first disease considered. A third disease is pseudo-hypertrophic paralysis, in which a fatty deposit in the muscle conceals the real atrophy in progress; a disease of childhood, but not due to any spinal affection; a disease of the muscular tissue itself. And lastly, he drew the picture of multiple neuritis, a disease due to degenerative processes in the nerves, independent of spinal lesion; a disease characterized by atrophic paral-

[1] Leyden: Charité Annalen, 1880.

[1] Leyden: Zeitsch. f. klin. Med., 1880.

[2] Leyden: Zeitschrift für klin. Med., I. Ueber Poliomyelitis und Neuritis.

ysis, associated with marked sensory symptoms, and with tenderness of the nerve, by a typical course, and, usually, by a favorable termination. By this article Leyden established the status of multiple neuritis as a distinct disease. Its symptoms were analyzed. Its lesion was described. The pathological stage in the progress of its discovery was complete.

When the characteristic features of a new disease have once been clearly pointed out, it is remarkable to observe how rapidly cases of it begin to be recognized. In the two or three years which followed the appearance of Leyden's article, numerous cases of multiple neuritis were reported in the journals. And many physicians, reviewing their records, recognized, in cases previously obscure or imperfectly diagnosticated, typical pictures of the new disease. It may be well to consider a few of these cases in order to complete our clinical knowledge of the affection.

Observation VI.—A female, aged thirty, of intemperate habits, but otherwise in good health, after suffering from formication, coldness, and pains in her feet and legs for some months, noticed an œdema of both legs. This increased rapidly after a few days, and the swollen limbs became painful to touch or pressure, and were the seat of severe lancinating pains, which were worse at night. Within a month the same symptoms appeared in the arms and hands, and a marked hyperæsthesia developed in all the extremities, as well as a rapidly progressing paralysis; so that, on admission to the hospital, six weeks after the appearance of the œdema, it was impossible for her to lift her limbs from the bed, or to extend her hands and fingers. The movements in the distal portions of all the extremities were much more impaired than those near the trunk, and in the paralyzed extensor muscles the faradic excitability was almost abolished. Heart and kidneys normal. She had some fever, and was delirious at night. The symptoms increased rapidly; the paralysis became total; respiration became difficult; the heart rapid, and a week after admission to the hospital she died.

Autopsy: Tubercles in the lungs. Brain and cord were normal. Nerve-roots were normal. In the nerves of the extremities marked degeneration was found, especially in the radial and tibial nerves. By the side of a small number of empty sheaths were found fibres, whose myelin was segmented and in drops, separated by empty spaces. The axis-cylinders were indistinct. There was no increase in the nuclei of the sheath of Schwann.

In discussing the case Dr. Lancereaux made the diagnosis of alcoholic paralysis, assigning the lesion to the nerves, and differentiating it from a myelitis or a meningitis. In so doing he criticised the views of Wilks and Lockhardt Clarke, who still considered alcoholic paralysis as a central disease. He cited another case, very similar, of a female aged thirty-three, in which the symptoms were pains, hyperæsthesia, tenderness, paralysis, with atrophy in the extremities, but in which œdema was not so marked and came on quite late in the course of the case. The same lesions were found. To these he added two cases in which the patients manifested the same symptoms, but had never drank. They were both, however, sellers of varnish, and lived day and night in an atmosphere permeated by alcoholic vapor, from which he concludes that chronic alcoholic poisoning can occur by absorption through the lungs—a valuable observation, but hitherto not confirmed.[1]

The following case was reported by Grainger Stewart, together with two other cases which resulted in recovery.

Observation VII.—A male, aged thirty-one, noticed during August, 1880, a weakness of the legs, and during the following month a pain of a prickling character in the legs and feet. These increased in intensity, and in October a similar feeling came on in the fingers and hands, accompanied by loss of power and stiffness. When seen, in November, he had tingling pain in both legs from the knee to the back of the foot, with numbness and feeling of cold in the toes and plantar surfaces, so also in the hands, to a less extent. No girdle pain or formication. Sensibility to touch was diminished in the legs and hands. Transmission of impressions was delayed. Sensibility to heat, tickling, and pain were all diminished, as was also the muscular sense in the feet. There was no nystagmus, although he complained of things dancing before his eyes. Sight was normal. There was no incontinence of urine or feces. The skin reflexes were absent in the soles, but normal in the abdomen and groins. The knee-jerk was absent. Voluntary motion was greatly impaired in legs and arms, and attempts to use the muscles caused pain. Electric irritability of the muscles and sensibility of the skin were much diminished. There were no vasomotor or trophic changes. His mental condition became changed during his stay, his memory was impaired, and he seemed drowsy. A month after his first examination he died of pneumonia.

Autopsy by Dr. D. J. Hamilton: The median, ulnar, and tibial nerves showed great changes. With a low power of the microscope the bundles of fibres appeared to be affected by fatty degeneration. With a high power it was found that the axis-cylinders were swollen so as to form a number of fusiform bodies in the course of the nerve-tubes. These at parts were divided into a number of round homogeneous colloid bodies. When set free these bodies underwent a fatty degeneration, forming compound granular corpuscles. In some fibres the axis-cylinder was totally destroyed, nothing but a quantity of fibrous tissue remaining. The cords of the brachial plexus and the sciatic nerves were normal. Slight evidence of secondary sclerosis in the spinal cord was found in the columns of Goll and in the direct cerebellar columns. Its origin could not be explained.[2]

It must not be supposed, from the fact that all the cases so far cited were fatal, that death is always the result in multiple neuritis. This is very far from the truth, and probably, if the mortality had been greater, the pathology of the disease would not so long have eluded search. The fatal cases have been brought together in order that the pathological appearances observed might be noted and compared, and might become somewhat familiar by repetition. It is time to enter upon the more careful study of their pathology, and so, for a time, let us leave the clinical features of the disease.

It may not be out of place, before proceeding to discuss the changes occurring in inflammations of the nerves, to review the normal anatomy of a nerve.

[1] E. Lancereaux: De la Paralysie alcoolique. Gaz. Heb. de Méd., 1881, p. 120.

[2] Grainger Stewart: Edinburgh Medical Journal, April, 1881.

The Histology of a Nerve-Fibre.—When a nerve-trunk is dissected, the connective-tissue sheath or perineurium enclosing its fibres torn away, and the individual fibres set free by teasing from the finer connective-tissue strands or endoneurium which bind them together, it is possible to distinguish certain parts, by means of appropriate methods of staining.

There is *first* the axis-cylinder, Fig. 1 (*a*). This is made up of a number of primitive fibrils arranged longitudinally and continuous throughout the length of the nerve. The fibrils are cemented together by a substance which appears finely granular. Each cylinder is supposed to represent a prolongation of a single nerve-cell; but whether the individual fibrils of which it is made up come from the same cell or from different cells, or from the fibrillary network in the central organ surrounding the cell, is a matter of hypothesis.[1]

There is, *secondly*, the myelin sheath surrounding the axis-cylinder, Fig. 1 (*b*). This is not a continuous tube, but consists of a series of little tubes, or interannular segments placed end to end. The point of junction of two adjacent segments is indicated by a constriction in the contour of the nerve-fibre, and if the myelin be stained with osmic acid it is seen to be deficient at these constrictions. If, by any means, the nerve-fibre be broken, the myelin in any segment will run out and collect in drops, showing that it is a semi-fluid substance, of fatty nature.[2]

There is, *thirdly*, a connective-tissue membrane surrounding the myelin sheath, the sheath of Schwann, Fig. 1 (*c*). This, like the last, is made up of segments cemented to one another at the point of constriction of the fibre. This constriction is known as the ring or node of Ranvier, Fig. 1 (*d*), and at its situation the sheath of Schwann is the only covering of the axis-cylinder. On the inner side of this sheath, and halfway between two nodes, is found a nucleus (Fig. 1, *f*). The sheath and nodes are made evident by staining with nitrate of silver, while the nucleus is seen best by staining with carmine or acid fuchsin. The mutual relation of the sheath of Schwann and the myelin sheath, are best understood by the study of their development, segment by segment. Ranvier[1] likens the production of any one segment of these sheaths to the production of a fat cell from a nucleated cell. Every nucleated cell is surrounded by a layer of protoplasm. As the fat forms, it collects within the layer of protoplasm in the vicinity of the nucleus, in the form of drops which finally unite into a mass. This mass is surrounded by a very thin layer of protoplasm and by the cell-membrane. On one side of it is seen the nucleus flattened out against the inner side of the membrane in the layer of protoplasm. Now, if each segment of the sheaths corresponds to the fat cell, and the myelin to the fat, the structure of the segment is clear. The membrane of the fat cell is represented by the sheath of Schwann. At the point of constriction of the nerve-fibre this membrane is cemented to that of the adjoining segment. Beneath the membrane, and flattened against its side, is the nucleus of the segment, lying in a layer of protoplasm in the middle of the segment. This layer of protoplasm forms a lining everywhere to the sheath of Schwann. With that sheath it is reflected around the axis-cylinder. Within the lining, in a state of semi-fluidity, is contained the myelin, which will run out if the limiting membrane is broken. By appropriate staining fine lines can be seen passing between the outer and inner layers of protoplasm, through the myelin sheath, the so-called incisions of Schmitt, Fig. 1 (*e*). These have been considered little trabeculæ of protoplasm within the segment in the midst of which the myelin lies. Recent staining methods seem to indicate that they belong to the sheath of Schwann and are connective tissue. The layer of protoplasm lying against the axis-cylinder, is the layer which was formerly described as the sheath of Mauthner. Some authorities consider that a layer of connective tissue similar to the sheath of Schwann surrounds the axis-cylinder, but this is still uncertain. Thus the nerve-fibre consists of a central conducting strand, surrounded and insulated by a series of tube-like segments of fluid contained within a membrane, which are joined to one another, forming a double protecting sheath.

Individual fibres are associated in bundles held together by fine connective-tissue cells whose nuclei

FIG. 1.

A Normal Nerve-fibre. *a*, Axis-cylinder; *b*, medullary sheath; *c*, sheath of Schwann; *d*, node of Ranvier; *e*, incisure of Schmitt; *f*, nucleus of the sheath of Schwann.

[1] Whatever their origin, it is certain that they pass together in the axis-cylinder to the periphery, and that there the axis-cylinder as such terminates, while the individual fibrils branch out in various directions, and, joining with other fibrils from other cylinders, form a fine plexus within the organ to which the nerve as a whole has gone. It has been thought possible to trace individual fibrils of the plexus into individual epithelial cells. Such is their termination in various internal organs, and in the skin. This is not, however, the only manner of termination of the axis-cylinder, for individual nerves can be traced directly to terminal organs, such as the terminal plates upon the muscle, and the terminal bulbs and corpuscles in the skin; in which cases no division or branching of the fibrillary constituents of the axis-cylinder has been discovered. The termination in each of the organs of special sense is still different. The axis-cylinder can best be seen by the acid fuchsin stain.

[2] The myelin sheath is not a necessary constituent of all nerve-fibres, for the majority of the nerves of the sympathetic system are devoid of such sheaths. Nor does the myelin sheath of the nerves of the cerebro-spinal system extend from end to end of the axis-cylinder. For the axis-cylinder first receives its sheath at some little distance from the cell from which it issues, and at its termination, where it breaks up into branching fibrils, the myelin envelope ceases. Throughout the course of the nerve, however, the myelin sheath is present in the cerebro-spinal nerves, forming a protecting envelope, and probably acting as an insulating substance as well.

[1] Ranvier: Leçons sur l'Histologie du Système Nerveux, tome i. p. 115.

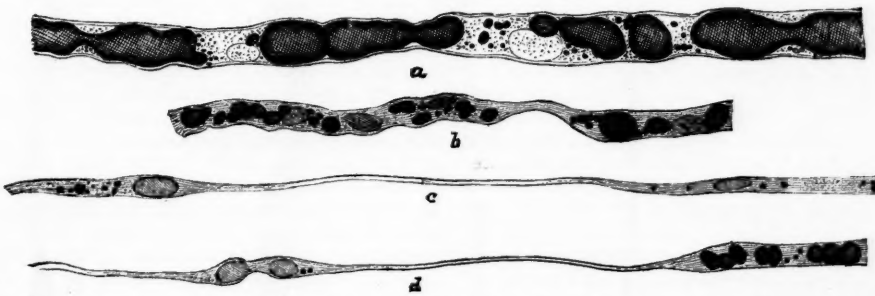
can be seen in a carmine or fuchsin stained preparation, lying always adjacent to, but outside of, the sheath of Schwann.[1] This has been called the endoneurium, while the connective-tissue sheath surrounding the entire bundle is named the perineurium. Capillary vessels with free anastomoses run within the nerve, their walls lying adjacent to the individual fibres, and thus affording a perfect nutrition.[2] Lymph-spaces also have been demonstrated within the nerve sheath, but not among the fibres.[3] That the interfibrillary spaces of the endoneurium, however, open into these perifascicular lymph-spaces is probable, from the fact that they do so in other organs. While it is evident that the nutrition of the axis-cylinder is derived from the circulatory fluids, it is probable that it is only at the nodes of Ranvier that the absorption takes place, since elsewhere the myelin sheath interferes with osmosis. Thus, if the nerve be put in nitrate of silver, it is only opposite the nodes that the axis-cylinder becomes stained.

The Pathology of Multiple Neuritis.—In studying the pathological processes which occur in multiple neuritis, it is necessary to keep these various elements of the normal nerve in mind, since each element is subject to changes. The exact character of these changes is best understood by observing the results of nerve-degeneration artificially produced in animals. And a study of this will not be out of place here, for, as we shall see, the changes occurring in multiple neuritis correspond quite exactly to those produced by experimental degeneration.

In considering the pathology of degenerative neuritis we enter at once upon a mass of controversial statements. It would seem to be a simple matter to establish, by observations upon nerves which had been experimentally compressed or severed, the changes which ensue in nerve injuries. But, as a matter of fact, there is, perhaps, no field of experimental pathological inquiry in which the results have differed more widely. In the first place, it is probable that the rapidity and even the character of the changes differ in different animals. Secondly, various methods of investigation, of hardening, dissecting, and staining the nerves, seem to have resulted in the production of different appearances. And, lastly, it is by no means certain that a uniform pathological process goes on after experimental lesions.

After a lesion of a nerve-trunk, a process of degeneration sets in at the point of injury, and involves a small portion of the central end and the entire peripheral part of the nerve, from the seat of injury onward. This process may be more or less complete, and may, or may not, be followed by a second process of regeneration in the diseased nerve. It is necessary to distinguish between the degenerative and regenerative processes; and, inasmuch as it is affirmed that they may proceed simultaneously in various parts of the same fibre,[1] it is not strange that the confounding of the two should have increased the confusion in the statements.

FIG. 2.



The Process of Degeneration (after Ranvier). *a*, Segmentation of myelin and axis-cylinder, with increase of protoplasm; *b*, disintegration of myelin into drops, increase of nuclei, partial absorption of débris; *c*, complete absorption of the disintegrated mass, leaving the Schwann sheath empty, or containing only débris and nuclei; *d*, the same, nuclei alone remaining.

In early times it seems to have been a question whether the destruction of a nerve was ever recovered from, and while the majority of authorities rightly contended that this was possible, there were those who denied it vehemently. Steinneck,[2] in 1838, seems to have determined this fully in the affirmative, using both the physiological and anatomical data at his disposal to good advantage. The exact processes of degeneration were first carefully studied by Nasse[3] and Waller,[4] and their results having attracted attention to the phenomena in question, a host of investigators have followed their lead. The most careful examinations of the entire series

[1] These connective-tissue cells which lie between individual fibres, have been recently the subject of study by Adamkiewicz (Adamkiewicz: Ueber Rückenmarkschwindsucht. Sitzungsbericht der Wiener Acad., Bd. 91, Abth. II.), and Rosenheim (Rosenheim: Arch. für Psych., xvii. S. 820). The former regarded them as nerve-corpuscles, but the latter has proven that they are true connective-tissue cells. They are best seen in specimens stained with a double staining of anilin gentian-violet, and methyl-blue, though they can be seen distinctly in fuchsin stained preparations, as has recently been demonstrated by Dr. Van Giesen, of the Laboratory of the College of Physicians and Surgeons. They appear on nerves after the age of five, and increase in number as the individual grows older, so that in elderly persons they are very numerous. They are round or spindle-shaped, and frequently appear to have little thorn-like processes. In addition to these connective-tissue cells there are fine fibrils of connective tissue, and bands of tissue lying between the bundles of nerve fibres.

[2] Ranvier, l. c., p. 253.

[3] Ranvier, l. c., p. 258.

[1] E. Neumann: Ueber De- und Regeneration der Nerven, Arch. f. Mikro. Anat., xviii.

[2] Schmidt's Jahrbuch., Bd. 26, S. 102, 1840.

[3] Müller's Archiv, 1839.

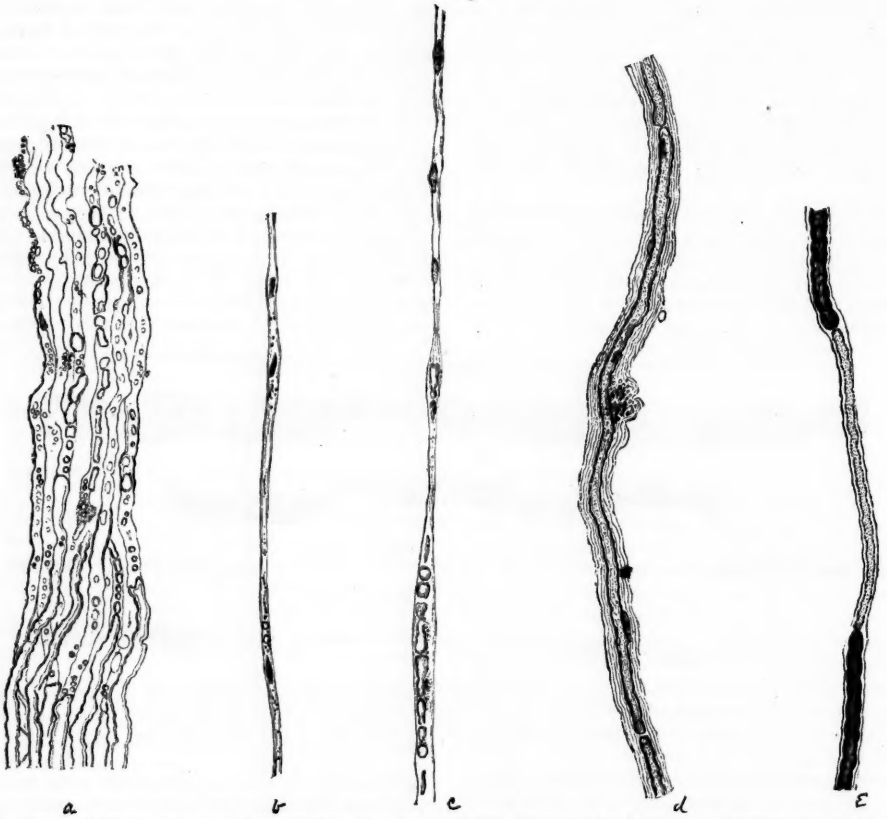
[4] Comptes rendus, 1852, vol. 34, p. 675.

of changes which go on in degeneration and regeneration have been made by Ranvier,[1] Phillipeau and Vulpian,[2] and Dejerine,[3] in France; by E. Neumann,[4] S. Mayer,[5] and Wolberg,[6] in Germany; by Hanken,[7] in Holland; by Schiff[8] and Tizzoni,[9] in Italy, and by Weir Mitchell,[10] in America.

The majority of writers upon nervous diseases, and upon general pathology, seem to have followed Ranvier closely, without any mention of the fact that other authorities differ from his conclusions. It will be necessary here to present the various views which are held.

The Process of Degeneration.— When a nerve is compressed by a ligature, or forceps without sufficient force to rupture the sheath of Schwann, the myelin is driven away from the point of pressure in both directions, and the axis-cylinder is disintegrated and mingled with it. It might be supposed that the nodes of Ranvier would prevent such a driving back of the myelin, but they seem to offer but feeble resistance, so that the entire fibre on either side of the compressed spot is bulged out for some little distance, the sheath of Schwann between the distended portions being left empty, or containing only a little granular débris. In a short time changes of a degenerative character are observed on both sides of the point of compression. Those on the central side are limited to the immediate neighborhood of the injured spot, and, according to Ranvier, do not affect the nerve for a distance greater than a centimetre from the point of compression.[1]

FIG. 3.



The Process of Degeneration and Regeneration going on in Normal Nerves (after Mayer). *a*, Segmentation of the myelin; *b*, contents of sheath of Schwann reduced to granular mass; *c*, sheath of Schwann, containing only débris and nuclei; *d*, granular mass in the sheath in process of regeneration; *e*, regeneration of a single segment after degeneration.

[1] Leçons sur l'Histologie du Système Nerveux.
[2] Gaz. Méd. de Paris, 1859; Comptes rendus à la Soc. de Biol.
[3] Arch. de Physiol., 1875, p. 567.
[4] Arch. f. Pathol. Anat., xviii. 302, 1880.
[5] Zeitsch. f. Heilkunde, ii., 1881.
[6] Deut. Zeitsch. f. Chirurgie, xviii. u. xix., 1883
[7] Internat. Monatsch. f. Anat. und Histologie, Bd. iii., 1886; Fortschritte de Medicin, Dec. 21, 1886,
[8] Comptes rendus, 1854, vol. xxxviii. p. 448; and Zeitsch. f. Wissen. Zool., 1856, vii. p. 145.
[9] Centralbl. f. Med. Wissen., 1878, Nr. 13.
[10] Injuries of Nerves, 1872.

[1] Some recent investigations of Friedländer and Krause throw doubt upon this statement. (Fortschritte der Medicin, Dec. 1886, They have discovered a considerable degree of atrophy in the nerve-trunks in the stumps of amputated extremities, and they traced this atrophy upward through the nerves, and into the pos-

On the peripheral side of the point of pressure the degeneration is extensive, involving the entire nerve down to its finest terminations. The process is held by some to be a gradually advancing one along the fibre from the point of pressure outward, while others believe that it begins at once in the entire length of the nerve. The first change noticed is a breaking up of the myelin sheath into segments, and then into smaller masses and drops (Fig. 2, *a*, *b*), which finally undergo further disintegration, either by a fatty or albuminoid degeneration, or by a process of saponification, until a finely granular mass alone remains. Tizzoni states that this process is partly due to the activity of migratory white blood-globules, a view which Ranvier supports, and which Neumann and Mayer deny. If, as seems probable, the connective-tissue cells of Rosenheim take an active part in all processes of nerve inflammation, it is possible that these are the bodies which Tizzoni mistook for leucocytes. Ranvier holds that the segmentation of the myelin is due to the increase of the protoplasm about the nucleus of the segment, and that it is this protoplasm which replaces the myelin. Neumann holds that the granular mass resulting from the disintegration is not protoplasmic, but is a débris capable of acting as the basis for processes of regeneration after undergoing a chemical change. All authorities admit that the granular mass may be gradually absorbed, leaving the sheath of Schwann collapsed and empty, or containing only granules of débris (Fig. 2, *c*).

As the myelin undergoes these changes the axis-cylinder usually becomes involved. Some authors, it is true, believe that it remains intact, and that, although deprived of its function, it is capable of resuming that function at any time when regeneration of the myelin sheath has taken place. Such authorities as Erb, Charcot, Weir Mitchell, and Wolberg have given their approval of this view. Ranvier believes that the protoplasm of the nucleus attacks and destroys the axis-cylinder as well as the myelin. Neumann holds that it is split up into segments like the myelin, becomes mingled with it, and undergoes the same process of chemical change, or absorption; and Mayer agrees with this view. (Fig. 3, *a*, *b*, *c*.)

The sheath of Schwann also takes part in the process of degeneration. When that process has fairly begun numerous nuclei are observed lining this sheath in each interannular segment. They may have come by a process of segmentation of the original nucleus of the segment, as Ranvier holds. But Neumann and Mayer have shown that they appear as early at the extremities of the segment as they do in the vicinity of the nucleus, and they therefore conclude that they are a free formation originating in the granular or protoplasmic mass. Tizzoni thinks them emigrated corpuscles. The most recent view of all is that of Rosenheim, that they come from the connective-tissue cells along the sheath which divide and multiply, and show powers of emigration as soon as the process of degeneration begins. When the granular mass is absorbed these nuclei remain scattered along the sheath of Schwann, and it has been suggested that, when in an empty sheath a new axis-cylinder appears, it owes its existence to these nuclei, which arrange themselves in a line, and develop into the new fibre (Wolberg). If no regeneration occurs they disappear gradually, and then the only relic of the former nerve-fibre is the empty, collapsed sheath of Schwann, which remains as a connective-tissue strand.

The increase of nuclei and connective-tissue fibrils in the endoneurium and perineurium which accompanies the process of degeneration, aids in the transformation of the nerve into a band of connective tissue.

The degeneration which affects the nerve is continued to the terminal plates upon the muscle, and these are changed into a mass of granules and finally absorbed, a connective-tissue plate being left.[1] Whether any changes occur in the sensory terminal organs, such as the tactile corpuscles or terminal bulbs, has never been ascertained. Those who believe that the individual axis-cylinder fibrils terminate in the epithelium of the skin may cite the trophic changes which often occur on the surface as evidence that this covering of the body shares in the nerve changes.

Authorities seem to agree that the same results follow a division of a nerve that are observed after its compression, with the difference that at the point of section the myelin runs out of the sheath of Schwann. The cut end of the nerve becomes swollen into a bulbous extremity by a growth of connective tissue.

Whether a true union of the divided ends ever occurs is still a matter of uncertainty. The majority of authorities, following Ranvier, affirm that while a primary coaptation of the ends by an exudate which is secondarily transformed into connective tissue, may occur and hold the ends in position, no true primary union of nerve-fibres is possible, and under all circumstances the degenerative process already described goes on to completion. Glück, however, claims to have observed an actual union of the two ends, with reëstablishment of function, at a time too early to have admitted the occurrences of degeneration and regeneration; and Wolberg, approaching the subject from the surgical side, and considering the results of nerve-suture, inclines to the same view.

In the midst of such contrary statements, what conclusion can be reached as to the actual facts? It seems evident that but one conclusion is certain, viz., that under different circumstances different processes occur. The various observers are equally trustworthy. It is impossible to choose one set of conclusions rather than another, or to rely wholly upon one series of experiments, however capable the observer may be, for they all rest upon repeated observations. It is undoubtedly

terior nerve-roots exclusively. They conclude, therefore, that the fibres affected are entirely sensory fibres. The axis-cylinder is only slightly affected by the atrophy, but the myelin sheath is wholly destroyed. Inasmuch as the degree of atrophy in a nerve-trunk was about the same, whether the amputation had been made near the body or near the end of the limb, they conclude that only certain of the sensory fibres degenerate upward. And as the tactile corpuscles and terminal bulbs are found only in the most distal parts of the extremities, it is suggested that only those sensory fibres degenerate which arise from these so-called tactile bodies. If this is proven to be true by further observation, the tactile bodies will assume the *rôle* of trophic centres for the sensory nerves. And in this case anything which interrupts the connection between the tactile bodies and the posterior spinal ganglion will produce a centripetal degeneration in the nerve. Hence in neuritis from section or pressure, as well as in multiple neuritis, such secondary degeneration is to be expected and should be looked for.

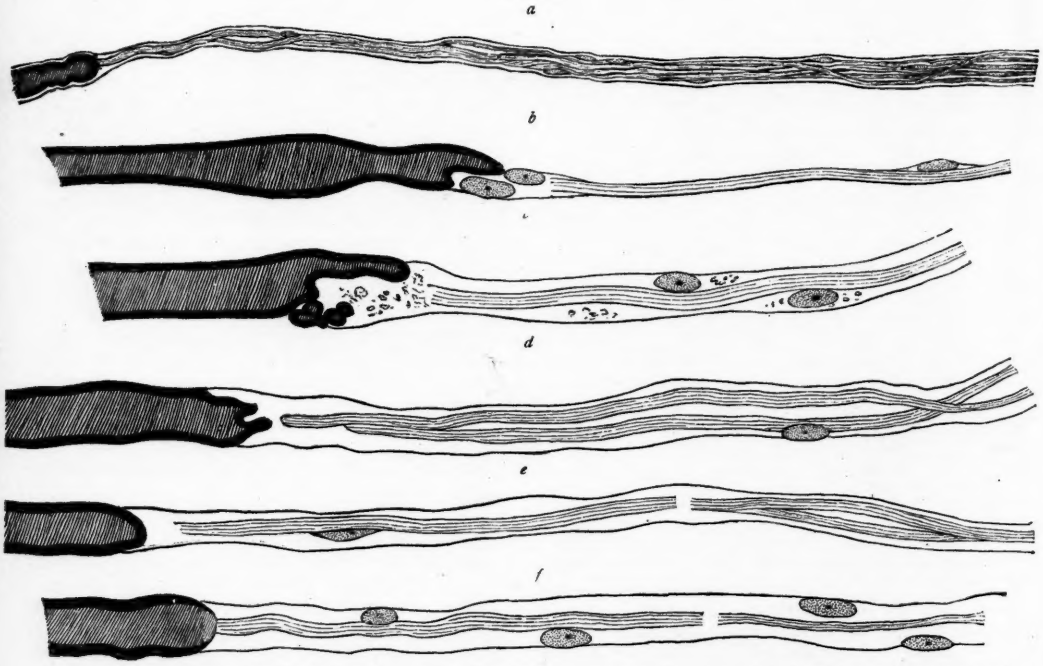
[1] Gessler: Die Motorische Endplatte und ihre Bedeutung für die Periphere-Lahmung. Leipzig, 1885.

true that in some cases the degenerative process, so graphically pictured by Ranvier, from the beginning segmentation of the myelin down to the final result in the connective-tissue strand, the relic of the empty sheath of Schwann, goes on; while, in other cases of a less serious nature, the destruction is less complete, and there remains a fibre consisting of a sheath of Schwann, containing a granular mass which may be either an axis-cylinder, or a mass capable of developing into an axis-cylinder under favorable circumstances. If this is the case, we can affirm that brilliant surgical successes, with rapid restoration of nerve function after suture, are possible when the partial degeneration is present, but are impossible when the total destruction of the nerve-fibre has occurred. And statistics show that there is a certain percentage of operations which fail of any result, though repeated on the same nerve.

widely divergent views are held. Ranvier[1] and his followers claim that the new nerve is wholly a product of the central end of the injured nerve, growing out from it and making its way along the track of the peripheral end, which takes no active part in the process. Neumann and Mayer, on the contrary, believe that the regeneration goes on in the peripheral end of the cut nerve, segment by segment being formed successively, beginning at the point of injury and proceeding outward, the entire nerve being built up by the union of each distal segment, with the one lying centrally to it, until this process has reached the end. These various views demand a more exact statement. (Fig. 4.)

Ranvier describes several ways in which the new fibres issue from the central end. He has seen the central end of an individual nerve-fibre become hypertrophied, and from this swollen part a single new fibre start out al-

FIG. 4.



Process of Regeneration (after Neumann). *a*, Outgrowth of new nerve-fibre from central end, division into numerous axis-cylinders (Ranvier); *b*, development of axis-cylinder in peripheral end, the new fibre separated from the old by nuclei; *c*, ditto, with increase of protoplasm destined to become a new myelin sheath, overlapping of old myelin upon the new; *d*, two new fibres developing in a peripheral segment and ready to unite with the central end; *e*, two segments nearly developed, the central one in advance of the peripheral one; *f*, final union of the peripheral with the central segment, nuclei still remaining.

It is evident that future research should be directed, not so much to determine which of the processes described occurs, as to settle under what circumstances the one is produced rather than the other. And such research will have an eminently practical bearing, inasmuch as it will also demonstrate under what circumstances nerve-suture is likely to be attended by success.

The Process of Regeneration.—After the process of degeneration has gone on for some time in the nerve-fibre, it may cease, and the process of regeneration may begin. With regard to the method of this process two ready medullated. He has also seen a single axis-cylinder grow out, and then divide into two, or even more, axis-cylinders, each of which develops into a complete medullated nerve. Or from the swollen central end of a single fibre several medullated and segmented fibres may originate, or one medullated fibre may be sent out with several non-medullated fibres coiled about it; or, lastly, a simple axis-cylinder branches out without a medullary sheath, divides into

[1] Loc. cit., ii. 42–67.

two, and each of these in turn divides into two, and so on, until the sheath of Schwann, which had originally but one axis-cylinder within it, contains as many as eight or more (Fig. 4, *a*). These new fibres appear like a brush upon the end of the divided nerve, and wind about each other, forming a sort of plexus. By the aid of the cicatricial tissue, which usually joins the central end with the degenerated peripheral end, the bushy, branching fibres are directed outward toward the periphery. And when they reach the peripheral end of the cut nerve they insinuate themselves into the old remaining sheaths of Schwann, or between those sheaths, and grow on and outward till at last they reach the termination of the peripheral end, and the regeneration is complete. The terminal plates upon the muscles are renewed by a production of protoplasm in the plate. The description given by Neumann and Mayer is very different. It will be remembered that they describe, as a result of the process of degeneration, a band of fibres each consisting of a sheath of Schwann containing a granular mass. The process of regeneration begins in this mass. Within it they have seen a narrow band of fine homogeneous substance appear, which has the structure of a rudimentary axis-cylinder (Fig. 4, *b*). This does not fill the sheath of Schwann, and is often pressed aside by the nuclei which lie in that sheath (Fig. 4, *c*). It is not at first continuous with the end of the old axis-cylinder, remaining in the central part of the compressed or divided nerve, but as it increases in definite structure it approaches this old axis-cylinder, and finally unites with it. At the point of union a ring of Ranvier is formed. As this axis-cylinder develops, a substance is gradually formed around it, which is stained by osmic acid. This increases in thickness as the protoplasmic mass and the nuclei diminish, until it finally forms a new myelin sheath. The new myelin sheath is never continuous with the old one in the central end of the nerve, since it is separated from that by the ring of Ranvier; but often at first the older sheath seems to bulge out and encircle the new sheath, though this appearance is never permanent (Fig. 4, *c*). At the point of union of the new fibre with the old one, nuclei are often found; but these, like the others, gradually disappear (Fig. 4, *b* and *d*). Lastly, a new sheath of Schwann is produced around the new myelin sheath, and within the old sheath of Schwann. It presses aside the old sheath, together with such masses of protoplasm, drops of myelin, and nuclei as may remain, leaving them thus wholly outside of the new-made fibre, so that they coalesce with, and make part of, the endoneurium. The new sheath of Schwann has but one nucleus in each segment, and presents the nodes of Ranvier at regular intervals.

Neumann has shown that this process goes on in every individual segment of the nerve-sheath, so that in segment by segment, proceeding toward the periphery, the regenerative changes occur, and as each segmen approaches completion it joins itself to the preceding one (Fig. 4, *e*), until finally the entire nerve is reëstablished in its entire length. As the degenerative process begins in the segment nearest to the point of compression, so does the process of regeneration; and in some nerves the two processes may be seen going on together, the segments near the seat of injury being renewed while those at the periphery are still in a state of degeneration. The new fibres do not grow out from the old ones, as Ranvier describes in the regeneration of cut fibres, but protoplasm, with specific developmental properties, forms and differentiates the elements of the new fibre, and then unites it to the old one. The new fibres are, at first, somewhat smaller in calibre than the old ones (Fig. 4, *f*), but they gradually attain a normal size, and then the process may be said to be completed.

While Neumann would have the new fibres develop from the granular mass remaining in the old sheath of Schwann, Gunther, Hjelt, and Weir Mitchell suppose that they may originate from the nuclei of the old sheath, or even from connective-tissue cells and neurilemma nuclei remaining in the connective-tissue strand, and to this opinion Wolberg agrees. It is evident, therefore, that the process of regeneration is by no means accurately determined. But here again it is possible that conflicting statements may be explained. The method of regeneration may, perhaps, depend upon the exact stage of degeneration reached before it begins. If the product of degeneration is a simple band of connective tissue, it seems probable that the nerve-fibre will have to grow into it from a central origin, as in its original development in fœtal life, unless the connective-tissue cells recently discovered are neuroplastic cells, and have the power of producing new nerves, just as cells of periosteum may produce a new bone. If, however, when degeneration ceases, there remains a sheath of Schwann containing a granular protoplasmic mass, it is not at all improbable that that mass may be differentiated into an axis-cylinder and a medullary sheath, and joined to the old nerve-fibre, a process which has its analogy in the medullation of nerves in the embryonal state. If we admit, with Wolberg, that in some cases the axis-cylinder is not destroyed, the formation of new myelin is a rapid matter. That some such process as the one last described must occur in certain cases is certain, when the rapid recovery after minor injuries is considered, and when the results of nerve suture are taken into account; for in both these conditions the return of function occurs long before a new nerve-fibre, starting out from the old one, could have reached the periphery.

It is affirmed by Mayer that individual nerve-fibres in normal nerves are constantly undergoing these processes of degeneration and regeneration, either because the necessary renewal of worn-out tissue takes place in this manner, or because slight injuries from pressure or over-strain are sufficient to start up degeneration in single fibres.

The process of degeneration in the nerves, consequent upon the destruction of the ganglion cells from which they arise (the so-called Wallerian degeneration) differs in no respect from that ensuing upon compression or division, excepting that in the latter case only the distal part of the divided nerve undergoes the pathological change, while in the former it is the entire nerve that is affected. And in the changes described in multiple neuritis the same progress of events and the same varieties of termination are observed.

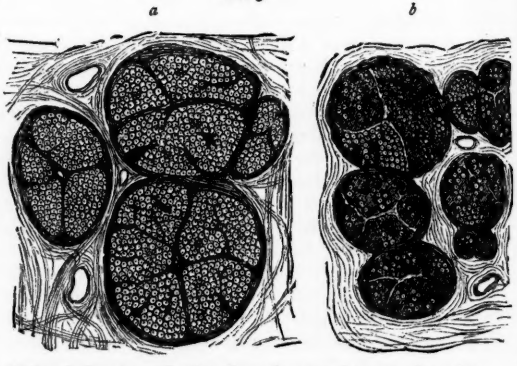
When the cases of multiple neuritis are examined from a pathological standpoint several varieties may be distinguished. In some of the cases, and these are the most numerous, there is a parenchymatous inflammation in the nerve-fibre only visible to the microscope.

At the outset of this inflammation the myelin sheath appears slightly swollen, is less homogeneous, and, from a difference of refractive power, is less translucent. It then becomes split up into segments of different length and form, the segmentation occurring preferably at the incisures of Schmitt, while the incisures at other parts disappear. Between these segments of myelin, a finely granular protoplasm is seen, in which new nuclei are found. These nuclei probably are the cells of Rosenheim which have emigrated, although a few near the nucleus of the sheath of Schwann may be due to its division. In some fibres the axis-cylinder may still be preserved. In others it is broken at the same places as the myelin. At the next stage of the process the changes are more marked. The myelin is now reduced to a series of small globules surrounded everywhere by granular protoplasm, and in this protoplasm the nuclei are now very numerous. The axis-cylinder cannot be distinguished in the mass, as a rule, but occasionally a fine line is seen passing through the mass, which may be a remaining cylinder. The succeeding stage presents a different picture. While up to this time the size of the nerve-fibre has remained about normal and uniform, it is now seen to vary. At places the fibre is still wide and filled with a granular mass, at other places it is narrow, the mass having disappeared, leaving either a collapsed sheath or a sheath containing only nuclei here and there. In a few such narrow fibres there seems to be an axis-cylinder lying directly within the sheath of Schwann, and occasionally separated from it at various places by nuclei. But this appearance is rarely seen. As a rule, no trace of the axis-cylinder remains. As any single fibre may show at some places constrictions, at others dilatations, the variation in its calibre is the most striking feature of this stage. In the terminal stage the calibre is uniform again, but is now everywhere reduced. The sheath of Schwann is empty, or contains only a little granular substance, and the nuclei are now less numerous than before. There is, in fact, only an atrophied tube with none of its original contents. These tubes lying side by side are folded and undulating, and appear like a strand of connective tissue.

These various stages of parenchymatous inflammation are to be seen in different fibres in the same specimen. Their appearance is identical with that observed in the course of degeneration of a nerve after compression, or after destruction of the spinal ganglion-cells. This has led such an accurate observer as Erb to advance the hypothesis that some slight changes in trophic cells in the spinal cord, not visible to the microscope, are present primarily, and that these changes are of a secondary nature.[1] But this cannot be admitted. Those who would explain the parenchymatous neuritis as secondary to changes in the cord, fail to explain why it should be limited, as it is, to the distal portion of the nerves, and not be continuous through the nerve-trunks, and anterior nerve roots into the anterior cells. They also offer no explanation for the cases in which the sensory, as well as the motor, nerves are affected, centrifugal degeneration of these nerves from spinal lesion being unknown. Strümpell, in meeting Erb's hypothesis, urges with reason that a parenchymatous neuritis has its parallel in other parenchymatous inflammations, and therefore does not need to be traced to any primary affection in trophic cells. He also mentions the existence of primary lateral sclerosis of the cord, similar in all respects to secondary degeneration of the pyramidal columns—yet now admitted to be a primary disease—as illustration of the fact that like changes are not always to be traced to one cause. It cannot but be admitted, therefore, that the first class of cases of neuritis must be considered as due to a primary parenchymatous inflammation in the nerve-fibres. This form seems to be much more frequent in its occurrence than the first variety, and constitutes the lesion in the majority of the cases hitherto reported. The appearance in section is seen in Fig. 5, *b*. The appearances of single fibres are the same as those shown in Figs. 2 and 3.

[1] Erb: Neurol. Centralbl., 1883, p. 481.

FIG. 5.



(After Joffroy.) *a*, Section through normal nerve; *b*, section through a degenerated nerve in a case of multiple neuritis, bundles reduced in size, individual fibres degenerating, their place taken by débris.

A second class of cases presents a different appearance.

In this class the mere inspection shows the nerve to have been the seat of pathological changes, for it is either congested, swollen, and lacking in lustre, or it is yellow and irregularly swelled by the accumulation of fat, or it is evidently reduced to a mere connective tissue strand. Upon teasing the nerve it is at once clear, from its brittleness, that individual fibres are lacking in continuity and are changed in structure. And if it is examined under the microscope the exudation of serum, and of lymphoid bodies, the great increase in the number of connective tisssue nuclei, the distended condition of the vessels, as well as the various appearances characteristic of nerve degeneration, are clearly seen. Here the inflammation is either originally an interstitial inflammation, or, more probably a diffuse one. It is possible that the degenerative processes in the nerves may have been due to the compression by the exuded products of inflammation within the nerve-sheath. In one or two cases where the patient died early in the disease, the nerve-fibres which lay near the vessels were affected to a greater degree than those lying deeper, and from this fact it was concluded that their degeneration was secondary. In other cases, however, all the fibres in a bundle are equally involved. Here it is probable that the inflammation was diffuse from the outset, paren-

chyma and interstitial tissue being affected simultaneously. The final result in either case is a diffuse inflammation. One marked feature in these cases is the large amount of fatty deposit found in the altered nerves. This is to be ascribed to the fact that myelin in undergoing degeneration breaks down into globules, and undergoes a fatty change. It is itself allied to fat, and making up, as it does, a large part of the bulk of the fibre, it would be noticeable in the product of degeneration if it were not absorbed. In this form the absorption seems to be interfered with by the vascular condition, and hence the residual amount of fat is increased. The changes observed in the nerve-fibres are very similar to those seen in the first form of neuritis, and need not be described again.

It is to be noted that in both of these forms the pathological changes are always more intense in, and are occasionally limited to, the peripheral terminations of the nerves. The nerve-trunks may be slightly involved in their distal portions, but it is very rare to find any changes in them at their origin from the plexuses. And, as a rule, the spinal nerve-roots in cases of multiple neuritis are normal. In making autopsies upon such cases this fact is to be remembered, and the nerves are to be removed down to their finer branches in the muscles and fascia.

One additional pathological form must be mentioned, since it has been described by such a careful observer as Gombault.[1] It is the so-called segmental periaxillary neuritis. In toxic neuritis from lead-poisoning, Gombault found that the degenerative process was not uniform in the entire length of the nerve-fibre. On the contrary, entirely normal segments alternated with the degenerated segments in the nerve. Mayer has noticed a somewhat similar condition (Fig. 3, *e*). The changes already described take place in the myelin sheath of the affected segment, even to its entire absorption, leaving the axis-cylinder in the sheath of Schwann. But as the adjacent segments are not involved, regeneration is thought to be more easily accomplished. Pitres and Vaillard[2] have noticed a somewhat similar condition in the neuritis occurring after diphtheria, although in their case the axis-cylinder as well as the myelin sheath was totally destroyed in many segments.

While it is, of course, impossible to describe definitely the process which goes on to complete regeneration of nerves in multiple neuritis, there is no reason to suppose that it differs in any way from that observed in experimental lesions.

The time required for the completion of the process will depend upon the severity and extent of the degeneration. When that is slight the recovery may be rapid, cases having been reported where a total cure took place in two months. As a rule, however, it is a slow process. The large majority of the cases on record required over six months for the complete regeneration, and in not a few cases from ten to sixteen months elapsed before the condition of the nerves was proved to be normal by the total disappearance of all symptoms.

With the end of 1883 we may consider the third stage of pathological discovery in the history of multiple neuritis as terminating. At that date the symptoms of the disease had been recognized as constituting a distinct clinical picture; hypothetical lesions in the spinal cord had been abandoned, and the exact pathology of the disease had been ascertained. Since 1883 the final period in the development of knowledge of the affection has been in progress. About one hundred cases, of varying degrees of severity, have been observed (see bibliography). The lesions described have been confirmed in all their details. The symptoms arising in the course of the disease have been subjected to careful analysis, together with their varied modes of combination. Certain diseases hitherto considered of spinal origin, are found to be due to peripheral disease. Thus it has been discovered that a form of ataxia may occur from multiple neuritis of alcoholic or arsenical poisoning, which closely resembles and was formerly confounded with tabes; also, that some cases supposed to be anterior poliomyelitis must now be differently named. And as physicians review their records, they find that former diagnoses require revision in the light of new discoveries, and that greater circumspection is to be exercised in differentiating spinal from peripheral affections. The grounds for such differential diagnosis will require attention in the next lecture. And, what is perhaps of greater importance, the fact has been elicited that some combinations of symptoms formerly supposed to be without a pathological basis, some of the so-called peripheral neuroses, really belong to this class of diseases. At present we have only time to allude to one or two forms of peripheral neuroses which must be removed from that unsatisfactory category of disease and be considered as peripheral neuritis.

And the first of these is the affection termed numb-fingers. This was first described by J. J. Putnam, of Boston, but met with instant recognition from neurologists, and from general practitioners all over the world. It is a disease chiefly seen in women between the ages of forty and sixty, usually associated with dyspeptic or uterine symptoms, but entirely independent of them. It begins as a tingling sensation in the ends of the fingers, felt at night, and sufficiently annoying to keep the patient awake. It then extends to the entire fingers, and may invade the hand, and is felt by day as well as by night. The fingers are so numb that all finer acts become impossible; the patient can no longer sew or knit, cannot be sure of holding anything securely, and finds herself unable to perform any delicate movement. Sometimes a slight degree of anæsthesia and analgesia can be discovered by ordinary tests, but often the disturbance of sensation is purely subjective. There is rarely any incoördination, and paresis is usually wanting. It may develop in the feet as well as in the hands, making walking more or less disagreeable, and adding to the discomfort of the patient. The affection is of indefinite duration; often subsiding quickly under treatment, sometimes baffling all attempts to arrest it. For a time it was considered a purely functional affection, then a spinal cord disease; but now, in the light of the parallelism between the symptoms mentioned and those which are characteristic of multiple neuritis, we cannot but consider it a slight form of this disease.[1]

[1] Gombault: Archives de Phys., 1873, p. 592; also Arch. de Neurol., i, 1.

[2] Pitres et Vaillard: De la Névrite segmentaire, Arch. de Neurol., xi. 337.

[1] Ormerod: St. Bart. Hosp. Rep., 1883, Sinkler: Phil. Med. Times, 1885.

The second of the peripheral neuroses which must be referred to peripheral neuritis is intermittent paralysis. Cases of sudden paraplegia, lasting a few hours and passing off as rapidly as they appeared, have been observed too frequently to admit of any doubt. Westphal has described such a case in which no cause could be found. Gibney reported some cases presumably due to malaria. Others have thought the disease of functional character, either central or peripheral. But in the light of recent observations upon infectious cases of multiple neuritis it becomes evident that these sudden, transient paraplegiæ find their adequate explanation in such an affection.

And, lastly, there are numerous cases of indefinite nervous symptoms, pain of various kinds, formication, and odd sensations grouped under the indefinite term numbness, flashes of cold and heat accompanied by actual changes in the temperature of the part, or only by apparent vascular irregularities, slight spasms, or tremors; functional weakness, with sense of fatigue not reaching the grade of paresis, and many equally obscure manifestations of disturbed function in various parts of the body, which receive their best explanation in the theory of multiple neuritis.[1]

Since it was one of the objects of the founder of this lectureship to determine the true nature of such peripheral neuroses, it is with the greater interest that we examine the disease to which they must be assigned.

In the present stage of progress in the history of neuritis much attention is being given to the etiology of the disease. Cases which, from their causation, were formerly separated, are now found to be closely allied in their pathology. Thus the forms of paralysis occurring after the ingestion of various poisons, such as arsenic, lead, and alcohol, are known to be due to a common pathological change. The various kinds of sensory and motor disturbance occurring as complications of the acute diseases, diphtheria, variola, typhoid and typhus fevers, and severe malarial fever, are traced to a lesion in the peripheral nerves. Tuberculosis is known to predispose to neuritis, and many cases formerly supposed, without question, to be produced by central or meningeal affections of a tubercular character, are now assigned to a peripheral cause. It is a question whether syphilis will cause a simple degenerative neuritis, but syphilitic affections of the nerves are easily recognized and well known. Nor can the nervous system escape the action of those microörganisms which are now recognized as the constant cause of many diseases. There is an epidemic form of multiple neuritis, fortunately not prevalent in this country, but occasionally imported here in the form of sporadic cases, known as kakke, or beri-beri, the bacillus of which has recently been discovered and cultivated. And, lastly, there is a class of cases, of supposed spontaneous origin, in which cold or overexertion are assigned as causes, but which need further investigation in regard to their etiology.

It is evident from this array of causes that several conditions formerly separated from one another because of the different circumstances of their occurrence are really forms of the same disease. But while they may be brought together upon a pathological basis, and while all have many symptoms in common, each of the forms of neuritis presents certain distinct features. Without dwelling too long upon any single variety, it will be necessary to review the different classes of cases in order to obtain a clinical picture of each. And such a review, together with the analysis of the symptoms of multiple neuritis, the discussion of its diagnosis from other diseases, its prognosis, and the treatment, will occupy us at the next lecture.

[1] Acroneuroses, Dana. N. Y. Med. Record, July, 1885.

ORIGINAL ARTICLES.

A NEW METHOD OF TREATING CHRONIC AURAL CATARRH.

By RALPH W. SEISS, M.D.,
INSTRUCTOR IN PATHOLOGY AND CHIEF OF EAR CLINIC IN THE PHILADELPHIA POLYCLINIC.

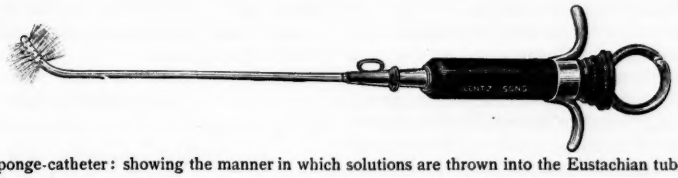
WHILE engaged in the study of the pathology of naso-aural catarrh during November and December, 1885, my attention was particularly drawn to the persistency of certain conditions of the pharyngeal mouth of the Eustachian tubes, as important factors in the general disease. Three principal forms of inflammatory disease of the tubes were noted: 1st. A condition of turgescence and inflammatory infiltration, the tubal lips appearing red and swollen, and secretion from the tubal glands being very active; the nasal pathological condition in these cases was hypertrophic catarrh in the stage of chronic venous distention with infiltration. 2d. The condition here noted accompanied advanced hypertrophic nasal catarrh, the turbinated bodies being largely converted into fibrous tissue. The prominence at the mouth of the tube was here found to be hypertrophied, the color was a dull reddish-yellow on the inferior and bright red on the superior surface. The pharyngeal tonsil was found to be enlarged, and the posterior nares more or less blocked with posterior hypertrophies. In these cases the mouth of the tube was frequently found completely filled with tenacious mucus. 3d. The third condition noted accompanied atrophic rhinitis, and here the lips of the tube were shrunken, the mucous membrane undergoing sclerosis, and the tubal mouth was often blocked by dried crusts.

It was found that after prolonged treatment of the nasal affection had partially or wholly cured the intra-nasal disease, that although the inflammatory conditions present in the Eustachian tubes might be improved, they were rarely if ever cured, the disease persisting in the last half inch of the tube.

Applications made directly to the tubal mouth with the post-nasal applicator or atomizer also gave but slightly better results. Various methods of treatment were tried without avail: the older methods of injecting solutions through the ordinary Eustachian catheter into the canal and middle ear were not tried, the evil effects of these applications in causing acute otitis media and severe inflammation high up in the tube being now well demonstrated.

With the object of making applications to the tube alone without danger of the fluid being forced into the middle ear or high up in the tube, I have devised the following instrument. It consists of an Eustachian catheter closed at the point, and having

the sides of the curved portion perforated with numerous very minute openings. A small syringe is fitted with a metallic nozzle fitting on the ground joint principle into the end of the catheter. The instrument may be made of German silver, but is better made of pure silver. The best average diameter is that of an ordinary Eustachian catheter—three to four millimetres—and the length is about the same, from fourteen to sixteen centimetres. The instrument is introduced along the floor of the nasal fossa like the older form of catheter, and when the beak is fixed in the Eustachian tube, suitable solutions can be injected through it, so thoroughly washing out and medicating the lower part of the canal, without the possibility of any of the fluid entering the middle ear or ascending high up in the tube, the fluid being thrown wholly in a lateral direction through the minute perforations.



Sponge-catheter: showing the manner in which solutions are thrown into the Eustachian tube.

In the first class of cases, those accompanied by turgescence and infiltration of the tubal lips with hypersecretion, two drachms of either of the following solutions were injected through the syringe catheter. Dobell's solution of the following formula: ℞.—Acid. carbol. c. p. gr. jss; sodæ borat., sodæ bicarb. āā gr. ij; glycerinæ ♏x; aquæ dest. fʒj.—M. And a saturated solution of boric acid in water and glycerine, ♏xx to fʒj. This form of treatment has been used with especial care in six cases of chronic aural catarrh accompanied by deafness and tinnitus aurium, and in every case with very marked results; the turgescence of the tubal lips subsided, the secretion became less in amount, and the patients volunteered the statement that they felt better; hearing in all cases was improved—two to five feet improvement for voice—and the tinnitus either grew better or slowly faded away entirely. The injections were made twice a week, the nares and pharynx being meanwhile under appropriate treatment. The syringe catheter has been used as an adjunct to older forms of treatment in from twenty to thirty additional cases, of which full notes have not been kept, but in almost every instance better results were obtained than from the use of the older methods alone.

In the second class of cases either the boric acid solution or the following was used: ℞.—Zinci sulph. gr. ij; acid. boric. gr. x; glycerinæ, ♏xx; aquæ dest. q. s. ad. fʒj, twice a week, a single syringeful (fʒj) being injected into the lower end of each tube. Much less marked results were obtained in these cases, but yet far better than has been obtained by any other method previously used by us. The tubal mouth soon became free from sticky mucus, hearing improved slightly, and the distressing tinnitus aurium was reduced to a minimum. The "lop-sided" "stuffed-up" feeling and dull aural pain, of which these patients almost invariably complain, were entirely relieved in the five cases in which this treatment has been regularly tried. The injections were here continued from three to six weeks.

In the third class of patients, those suffering from atrophic catarrh, but little good was achieved by the use of the syringe catheter—and the same may be said of all forms of treatment of the *nasal* lesions in many cases of this disease. The solutions used in these cases were a Listerine (Lambert) solution of the strength of fʒjss to fʒviij of water, and in certain forms of sclerosis Boulton's solution, of which the following is the formula: ℞.—Tinct. iodini comp. ♏xx; ac. carbol. (cryst.), ♏vj; glycerinæ fʒvij; aq. dest. fʒv.—M., and place in water bath of 100° until the solution becomes colorless.

Some relief from tinnitus and slight though appreciable improvement in hearing were, however, obtained in some of these cases by the use of this instrument along with careful treatment of the throat and nares, the results exceeding our expectations.

We have also used this catheter in a number of cases of acute tubal catarrh following coryza, using solutions of cocaine muriate, morphine sulphate, and boric acid of various strengths, with marked benefit; and treatment by the syringe has done much good in the severe tubal inflammation which usually accompanies purulent otorrhœa, various solutions being used. Prof. C. H. Burnett, to whom I am indebted for many suggestions and favors while devising and testing this catheter, has used the instrument to a considerable extent in his private practice, and tells me he has had markedly good results in some cases of chronic catarrh in which it was regularly used.

Considerable care and experience are requisite to use the instrument properly; after carefully fixing the beak in the mouth of the tube, the solution must be injected without forcing the curved portion forcibly against either wall, and the moment the fluid is thrown in the catheter must be withdrawn, or the patient will be choked by the fluid deluging his larynx as soon as the palate is relaxed. Of course, the rules which govern the introduction of the ordinary Eustachian catheter must be observed here, no force whatever being used. In skilful hands we have never seen the application do any harm whatever, or cause any considerable pain.

Without claiming great things for this little instrument, a year's experience with it has demonstrated to us its great value in selected cases, and we think it may serve a good purpose in aural therapeutics.

The instrument is made by Chas. Lentz & Sons, 18 North Eleventh St., Phila.

49 NORTH 17TH STREET, PHILADELPHIA.

STEEL PIN IMPACTED IN LEFT BRONCHUS FOR TWENTY-THREE MONTHS; SPONTANEOUS EXPULSION AFTER TRACHEOTOMY; RECOVERY.

BY H. R. WHARTON, M.D.,
SURGEON TO THE CHILDREN'S HOSPITAL, INSTRUCTOR IN CLINICAL SURGERY IN THE UNIVERSITY OF PENNSYLVANIA, AND ASSISTANT SURGEON TO THE UNIVERSITY HOSPITAL.

ELLEN MCD., aged seven years, was admitted to the Children's Hospital July 3, 1884, with the following history: On July 1st she had swallowed a large glass-headed mourning pin, which act was immediately followed by a violent paroxysm of coughing and difficult breathing; both of these symptoms had continued and increased in severity up to the time of her admission into the hospital.

When I saw her a short time after her admission, she presented symptoms of dyspnœa of so urgent a character that I immediately performed tracheotomy, which relieved her dyspnœa, but a careful search of the trachea failed to reveal the presence of the foreign body. A tracheal tube was introduced and the case was carefully watched, but she had no further return of her symptoms of dyspnœa during seven weeks' stay in the hospital.

During this time her temperature was considerably elevated and she presented an area of dulness over the apex of the left lung posteriorly.

The tube was removed at the end of three weeks, and at this time the trachea and right and left bronchi were again explored to discover the presence of the foreign body, but without success. After this the wound healed promptly and the patient was discharged from the hospital. The patient was under observation in the out-patient department of the hospital for some time, but presented no further symptoms of respiratory obstruction, although she had at times a cough, and examination of the chest revealed dulness and moist râles over the upper portion of the left lung posteriorly.

In July, 1885, she had a sudden and severe attack of dyspnœa which passed off in a few hours; in this attack she was seen by Dr. Morris J. Lewis, one of the physicians to the hospital, who described her condition as very alarming.

She presented herself at the hospital a few days after this attack, and examination of the chest revealed the same conditions as are mentioned above.

In June, 1886, she had a severe paroxysm of dyspnœa and coughing, and expectorated a steel glass-headed pin one and seven-eighths inches in length, which had remained in her respiratory passages for twenty-three months. After this she had no further trouble, and the cough of which she had complained at times completely disappeared.

Remarks.—Although foreign bodies may remain impacted in the air-passages for a considerable period of time without producing alarming symptoms, and finally be expelled spontaneously, yet it cannot be denied that their presence may give rise to complications which will place the life of the patient in imminent danger, so that it is generally conceded that surgical procedures should be promptly adopted for their removal.

A point of interest in this case was the entire relief of the urgent dyspnœa afforded by the operation of tracheotomy, although the opening into the trachea was far above the site of the impaction of the foreign body, and I have seen the same relief afforded when the operation has been performed in diphtheritic cases, in which the trachea was found obstructed with membrane adherent to the trachea below the tracheal wound; and I think this can only be explained by the theory that in all these cases the dyspnœa is in a measure due to a reflex laryngeal spasm. The sudden and severe attack of dyspnœa occurring about a year after the introduction of the foreign body into the air-passages, was at the time supposed to be due to the change of position of the foreign body, which, in the light of recent developments, was probably the correct explanation, as the foreign body was subsequently expelled during an attack of this kind.

TREATMENT OF THREATENED RUPTURE OF THE UTERUS BY MANIPULATION AND POSTURE.

BY E. P. DAVIS, M.D.,
OF PHILADELPHIA.

THE following case will serve as a type of some of those in which this serious accident is threatened, and the mode of treatment was found conservative and efficient.

A. B., primipara, a well-formed brunette, aged twenty, had entered the first stage of labor. Abdominal palpation demonstrated that the fœtus occupied the right half of the uterus, its back at the mother's right; the feet in the right upper segment of the uterus; the heart sounds heard plainest on the right side below the umbilicus; the head at the symphysis pubis. Upon vaginal examination the frontal suture was found extending obliquely to the left sacro-iliac synchondrosis: the greater fontanelle lay in the centre. The os uteri was permeable for only two fingers. The inspection of the abdomen showed at the upper border of the lower uterine segment and upon the right a well-marked bulging; it was evident that the distention of the uterus at this point was excessive. Meconium was constantly escaping in small quantities; labor pains were regular, but of moderate strength; fœtal heart sounds were regular and strong.

The fœtal position was evidently strong cephalic extension, and it was probable that operative interference would be necessary; the danger most imminent was uterine rupture.

In the non-dilated condition of the os uteri it was determined to endeavor to secure rotation and spontaneous birth by posture and external manipulation. The patient was accordingly placed upon her left side; an attendant was ordered to sit beside her, and, by gentle manual pressure upon the abdominal projection, aid in rotation; the fœtal heart and the maternal temperature were carefully watched; the pressure made was gentle and intermittent. In less than three hours the tumor had become smaller, and vaginal examination showed an improvement in the position of the head. This improvement continued; the labor was tedious, but operative interference was

not necessary; and in about twelve hours from its beginning labor terminated normally, and the puerperal period was without complication.

In cases of this kind which occur where a trained attendant is not obtainable, a method employed by Betz, and described in the *Wiener med. chirurg. Centralblatt* for November 26, 1886, will commend itself. Betz used a sandbag weighing five or six pounds, which afforded continuous pressure from a broad and perfectly applied surface. This was laid upon the projection formed by the head, and its position changed from time to time as the sensations of the patient indicated. In five hours the natural forces effected rotation, and the labor ended spontaneously.

These simple methods of aiding flexion and rotation at a period of labor when operative interference is difficult and dangerous are certainly worthy of consideration, especially when so serious a complication as uterine rupture is threatened.

MEDICAL PROGRESS.

EFFECT OF FREEZING ON THE TYPHOID GERM.—DR. JOHN S. BILLINGS, U. S. A., writes as follows to the *Sanitary Engineer* of Jan. 29, 1887:

It is well known that freezing water does not destroy the vitality of all living organisms contained in it, although it does appear to kill some of them. To settle the question as to the effect of freezing on the bacillus of typhoid, I have had the following experiments made:

Jan. 10, 1887. Five cubic centimetres of sterilized water in a test-tube were inoculated with typhoid bacillus and exposed to the outer air during the following night at a temperature of about 10° F. It was found solidly frozen in the morning.

Jan. 11, 2 P.M. This frozen mass was thawed, and from it there were inoculated one agar and three gelatine tubes.

Jan. 13, 10 A.M. There is decided typical development of the typhoid bacillus in the agar tube and in two of the gelatine tubes.

Evidently, therefore, the vitality of the typhoid bacillus is not destroyed by freezing.

DRUMINE: A NEW LOCAL ANÆSTHETIC.—An alkaloid has lately been obtained from the plant, Euphorbia Drummondii, N.O. Euphorbiaceæ, by Dr. John Reid, of Port Germein, South Australia, which promises, if report be true, to compete with cocaine, as an agent for producing local anæsthesia. A tincture of the plant is made with rectified spirit, or proof spirit, acidulated with hydrochloric acid, and, after standing a few days, the spirit is distilled off, ammonia added in excess, and the whole filtered. The residue, after the smell of ammonia has disappeared, is dissolved in dilute hydrochloric acid, and filtered through animal charcoal, to destroy the coloring matter which is abundant and inactive. This filtrate is evaporated slowly, and leaves the alkaloid. It gives a colorless solution, with little taste. It is almost insoluble in ether, freely soluble in chloroform and water, and deposits from solution microscopic acicular and stellate crystals. The crystals deposited from the hydrochloric solution filtered through animal charcoal, are circular or boat-shaped. They are colorless, and seem to be less soluble in chloroform. Sheep and cattle are stated to die in great numbers annually, in consequence of having eaten this plant, the poisonous qualities of which vary in proportion to the quantity of milky juice present. Sheep, bullocks, and horses die in from twenty-four hours to seven days after eating it, all of them presenting paralysis of the extremities, some of them hanging the head as if tipsy; the appetite does not appear to be interfered with. It is curious that the animals avoid the weed at first, except under pressure of hunger, but once having partaken of it, they seek for it and eat it with avidity. Injection of a solution of the alkaloid into the nostrils of a cat, produced stupidity and indifference to stimuli, with a placid, stupid expression like that of an animal under the influence of a narcotic. The limbs appeared paretic. A few drops of a four per cent. solution dropped into the eyes of another cat produced insensibility to the extent of allowing the conjunctiva to be touched, and the orbicular muscle no longer contracted with the same vigor. The pupil was not appreciably dilated. Three grains were then injected subcutaneously, but, beyond local anæsthesia, no effect was noted. A larger dose by the mouth promptly produced paralysis of the limbs, and slow, difficult breathing. When dying, strychnine was injected, but failed to produce any muscular contractions. Ten minims of the 4 per cent. solution injected into the hind leg of a cat, seemed to produce paralysis of sensation, but not of motion. No convulsions ever followed its use. In the course of experiments on his own person, Dr. Reid found that the drug produced anæsthesia, with loss of taste when applied to the tongue or nostril; but small doses swallowed were not followed by any perceptible constitutional symptoms. He tried it subcutaneously in a case of confirmed sciatica in an old man, and the essay was followed by complete, and, so far, permanent relief; in sprains, it was very useful in relieving the pain. Dr. Reid recommends the use of the alkaloid in small operations, local irritation, and sprains.—*British Medical Journal*, Jan. 1, 1887.

FORMULÆ FOR LANOLINE OINTMENTS.—A compound of diachylon ointment and lanoline, useful in eczema, is as follows:

Unguent. plumbi,	
Lanolinæ	āā 50 parts.
Adipis	10 "

In seborrhœa:

Lanolinæ	℥jss.
Olei theobromæ,	
Adipis	āā ℈iv.
Tinct. benzoin.	℈ij.
Ætheris	gtt. ij.

Used as a pomade.

The following is a lanoline cream:

Spermaceti	10 parts.
Ol. olivæ	30 "
Lanolinæ	40 "
Aquæ	50 "

This is also useful in seborrhœa.

Wilkinson has used the following prescription successfully in scabies:

Sulphur. sublimat.,	
Picis liquid.	āā ʒij.
Lanolinæ,	
Sapo. virid.	āā ʒss.
Pumicis pulv.	gr. 75.

In the efflorescences of psoriasis, the following pomade is useful:

Chrysarobinæ	10 parts.
Lanolinæ	40 "
Adipis	10 "

—*Annales de Dermatalogie et de Syphiligraphie*, December 25, 1886.

ABSORPTION OF FAT IN ACUTE INTESTINAL CATARRH.—DR. VIAZHLINSKI, who has been working in Professor Ivanovski's laboratory of pathological anatomy in St. Petersburg, has published an interesting paper on the question of the absortion of fat in acute catarrh of the small intestine. The literature, to which he first alludes, comprises more than ninety papers, etc., including those published by Goodsir, Schäfer, and Watney in this country. His own observations were conducted by inducing acute intestinal catarrh in animals by means of irritant drugs, and then feeding them with milk or other fat-containing food, after which the animal was killed and the intestinal villi examined with the help of osmic acid, which he found the most satisfactory agent for making the preparations. The drugs used were colchicin and croton oil. Several animals (cats and dogs) died, so the quantity of colchicin was reduced from one-thirtieth to one-sixtieth or one-hundredth of a grain, which produced choleraic stools. Croton oil was given in repeated doses of five or six drops, producing diarrhœa with exhaustion. When the animal presented, besides diarrhœa, an inflated abdomen, constant borborygmi, and a considerable degree of exhaustion, an acute catarrh of the small intestine was considered to have been established, and the fatty food was administered. The microscopic observations showed that in catarrhal conditions, as well as in health, the fatty particles are absorbed by their inclusion within round or amœboid cells—*i. e.*, leucocytes—the differences in the diseased state being merely a quantitative one. No evidence could be obtained of any part being taken by the epithelial cells in the absorption of fat.—*Lancet*, Jan. 8, 1887.

PARAFFIN IN SURGERY.—It is perhaps not so well known in the profession as it ought to be that paraffin is a most useful material in surgery, in many cases superseding plaster of Paris. It answers admirably for splints, and for jackets for young children suffering from spinal disease. The advantages of it are that it is clean, light, is capable of being moulded while it is soft, and sets rapidly by pouring cold water on it. It must be procured with a melting point of 130°. When ordering it it is necessary to specify the melting point required. The mode of using it is this: The paraffin is melted by placing a tin can containing it in a pot of boiling water. A muslin bandage well sprinkled with iodoform is put on the limb, or other part to which the paraffin dressing is to be applied. Muslin bandages loosely rolled are allowed to soak in the paraffin for a few minutes. If much support is required a piece of cotton-wool is also soaked in the paraffin. This is placed where most stiffness is wanted, and the bandages applied as for plaster-of-Paris dressing. They may, however, be drawn tight as the paraffin shrinks from the limb in cooling. Cold water is then poured on it, and the limb held in position till the paraffin has set. When a bone tends to project, it can be kept in position by the finger till the paraffin has set. The hole left by the finger can then be filled up with some melted paraffin. In cases of compound fracture the paraffin has the great advantage of not being affected by the discharge from the wound.—*Provincial Medical Journal*, Jan. 1, 1887.

CALOMEL IN HEART DISEASES.—STITLER has added to the studies of Jendrássik on the diuretic effects of calomel, and reports his experiences with fourteen cases. The daily dose was from seven to nine grains, and was invariably followed by good results.

In the case of a man, aged sixty-five, suffering from mitral insufficiency and chronic endarteritis, the symptoms of hepatic congestion, general œdema, and dyspnœa were promptly relieved, and a progressive diuresis occurred which began with the passage of 30 ounces of urine daily, and increased until at one time a maximum of 100 ounces was reached. The diarrhœa caused by the calomel was moderated by opium.

Stitler observes that the drug should be given three or four days before the diuretic effect can be expected, and then it is prudent to suppress it when diuresis is fully established.

It is a valuable adjuvant to digitalis in the treatment of cardiac disease.—*Bulletin Général de Thérapeutique*, November 30, 1886.

THE TYPHOID BACILLUS.—MM. WIDAL and CHANTEMESSE have succeeded in carrying researches on the bacillus of typhoid rather further than Gaffky, who described it. The centre clear space is not, they say, characteristic, as Artaud supposed, for it is found in other bacilli, especially those of old cultures; and it is, they believe, the beginning of the death of the microbe. Spores are produced between 37° and 38° C. It does not liquefy gelatine, and is easily cultivated on potato. Gaffky was unable to find the bacillus in the living subject, or to inoculate it. MM. Widal and Chantemesse have found it during life by making a capillary puncture of the spleen, and they have been able to inoculate both mice and guinea-pigs so as to find the bacillus in the abdominal viscera and lungs. In a case where a typhoid patient aborted in the fourth month the bacillus was found in the placenta.—*Lancet*, January 15, 1887.

TROUBLES OF HEARING AMONG RAILWAY EMPLOYÉS.—LICHTENBERG, of Budapest, has examined the hearing of 250 railway employés, and found 92, 36.8 per cent., in the number who had impaired hearing: 32 of the cases were catarrhal affections, 3 were diseases of the labyrinth, and 30 cases were those of the external ear. These results show the prevalence of impairment of hearing among a class whose occupation exposes them to such diseases, and in whom the public are interested as occupying responsible positions.—*Revue Mensuelle de Laryngologie*, January 1, 1887.

THE MEDICAL NEWS.

A WEEKLY JOURNAL

OF MEDICAL SCIENCE.

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SATURDAY, FEBRUARY 5, 1887.

ANTIFEBRIN.

FROM the reports in our German exchanges we notice that this new remedy has received an extensive trial, and the results so far seem to indicate that another valuable antipyretic has been added to our resources. Antifebrin is a neutral body prepared by heating aniline with acetic acid, and, when purified by successive crystallizations, it forms a white, odorless powder with a sharp but not unpleasant taste. It is insoluble in cold water, but soluble in warm water, or alcoholic fluids.

CAHN and HEPP, who introduced the drug, have been studying its effects in Kussmaul's wards at Strassburg, and in Nos. 1 and 2 of *Berliner klin. Wochenschrift*, 1887, give a full account of their observations based upon its use in sixty cases. It is given in doses of from five to fifteen grains. Eight grains is the usual dose, and it may be administered in warm water, or in a little alcohol and water, or in capsule. In larger doses it is not poisonous, and from sixty to ninety grains have been taken in the day without any ill effects. Fever patients rarely require more than thirty grains a day in divided doses. It is best to give a single dose of eight grains, to be followed, if necessary, by smaller amounts, in order to keep down the temperature. The effect is usually manifest in an hour, and, as a rule, there is a reduction of from three to five degrees in as many hours. Sometimes the fall is more rapid, and within two hours there may be a drop of five or six degrees. Copious sweating is almost invariably associated with its action. Chills have not been observed. The drug is well borne by the stomach, and in no case caused nausea or vomiting. The duration of action is variable, but in the acute fevers, after four or five hours the temperature gradually rises again. The administration of smaller doses may check this tendency. In typhoid cases an improvement in the general condition was often noticed after its use, and the mind became clearer. In other instances the patients expressed themselves as more comfortable; and in no case was there the depression which is sometimes seen after the administration of antipyrin or thallin. The pulse is also reduced in frequency, and the secretion of urine increased. The authors doubt if antifebrin has any specific action in typhoid fever, but in acute rheumatism it seems to act like antipyrin, not only on the fever but also on the inflammation, reducing the swelling and relieving the pain.

We can confirm these observations on the use of antifebrin. We have found that it acts promptly in comparatively small doses, is easy to take, and is free from the unpleasant after-effects of some other antipyretics. It is a cheap drug, costing not half the price of antipyrin, and seems likely to prove a valuable addition to the pharmacopœia.

INTRAPERITONEAL RUPTURE OF THE BLADDER.

THE late Professor Gross, in 1851, suggested the propriety of opening the abdomen and sponging out the extravasated urine and blood in intraperitoneal rupture of the bladder, with the retention of a catheter in the organ; a suggestion which was successfully resorted to, in 1862, by Walter, of Pittsburg, and, subsequently, with fatal results, the bladder itself having been sutured, by Willett, Heath, and McGill. Hofmokl had a recovery after laparotomy and suture, but in his case the description does not indubitably show that the rent involved the peritoneal investment of the viscus.

The almost uniformly fatal issue of this rare accident, with or without surgical intervention, adds additional interest to two recoveries recorded in *The Lancet* for December 11, 1886, by SIR WILLIAM MACCORMAC. In both cases the belly was opened, the laceration closed, and the cavity of the peritoneum washed out with a one per cent. warm boric solution. In one the rent, four inches long, and extending from the superior fundus to the rectovesical cul-de-sac, was closed with sixteen points of Lembert's suture, made with fine carbolized silk, and additional catgut sutures fortified the line of union where closure seemed to be insufficient between the silk stitches. In the second case the laceration involved two inches of the upper and posterior part of the bladder, and was closed by twelve points of suture, carried, as in the first case, through the serous and muscular tunics. In one a glass drainage tube, carried into the rectovesical cul-de-sac, was allowed to remain for four days, and a soft catheter was retained in the bladder; but these con-

trivances were dispensed with in the second case, as indeed, they may always be, provided peritonitis is absent, and distention of the bladder with boric solution shows that it does not leak.

It need scarcely be said that it is very desirable to intervene at as early a date as possible when the bladder is ruptured; but before severe symptoms have set in, the recognition of the accident is not always easy. Hence, in the earlier period, instead of practising an exploratory laparotomy in doubtful cases, it will be better to resort to the simple expedient recently described by R. F. WEIR, in the *Medical Record* for January 22d. In a patient, seen by that surgeon six hours after a crush, the decision of intraperitoneal rupture was difficult to arrive at, but it was satisfactorily solved in the negative by distending the bladder. A rubber catheter having been passed into the viscus, a Petersen's bag was inserted into the rectum and filled with seven and a half ounces of water. The line of suprapubic dulness having next been outlined by a colored pencil, a similar amount of carbolized water was thrown into the bladder, through which the contour of the viscus could be plainly felt above the line of blood extravasation previously felt in the suprapubic region. The water was then allowed to escape, and measured, when it was found to correspond to the quantity thrown in. Hence, the demonstration of absence of laceration of the bladder was perfect. Weir urges that the distention of both the rectum and the bladder, in making this test, should not exceed seven or eight ounces.

MULTIPLE NEURITIS.

DR. ALLEN STARR has been particularly happy in his choice of a subject for the first course of the Middleton Goldsmith Lectures. Although it is only six years ago since Leyden established the status of multiple neuritis as a distinct disease, a very large number of facts have accumulated, and an attempt is here made to classify these facts, to weigh their importance, to draw logical conclusions from them, and to present these conclusions in a clear and accessible form. The historical sketch with which the lecture opens presents many points of interest, to which, had we space, we would gladly refer, but we must allude to the vivid description of alcoholic paralysis by that great New England physician, James Jackson, in 1822. As Dr. Starr remarks, it is scarcely possible, at the present day, to add to his account of the main features of this important form of multiple neuritis.

The elaborate discussion of the histology of nerves and of the processes involved in their degeneration and regeneration clears the way for a statement of the morbid anatomy of multiple neuritis. There is still much to be done before we possess a satisfactory anatomical picture of all the stages of parenchymatous inflammation in nerves. In many instances it appears identical with the degeneration which follows compression of a nerve, or destruction of the spinal ganglion cells, while in others it seems to be preceded by an acute diffuse neuritis, with swelling and exudation of serum and leucocytes. An important feature is the frequent limitation of these changes to the peripheral termination of the nerve, and in future investigations this should be carefully borne in mind.

The etiology of multiple neuritis is curiously associated with toxic agents of various kinds. Lead, arsenic, and alcohol are responsible for a large proportion of the cases. The acute specific diseases, diphtheria, smallpox, scarlet fever, measles, and typhoid fever, are not infrequently complicated with motor and sensory disturbances, which we now know to be due to lesions of the peripheral nerves. Syphilis is a doubtful cause. The accurate studies which have been made of that remarkable disease, beri-beri, have thrown important light on the etiology of peripheral neuritis, and the observations which indicate that there is a special bacillus associated with the disease are of special interest in connection with the nerve lesions in lepra, which are apparently induced by the development of parasites in the nerve-sheaths.

MYOSITIS OSSIFICANS.

THE occurrence of the development of bony tumors in the substance of the muscles is sufficiently rare to make it worth while to call attention to a case of this kind reported recently by SYMPSON, in the *British Medical Journal*. The patient was a boy seven years old, with a distinct family history of rheumatism, although he had never had this disease, and of an injury—a fall upon the right shoulder, followed by a painful swelling over the scapula of the same side, which subsided and was succeeded by a similar swelling over the other scapula. At the time of observation, March 27, 1885, the boy had no motion in the right shoulder-joint and but little in the left. There were elastic nodulated tumors over both scapulæ, and in the position of the long head of the right triceps. The tumors were tender on pressure.

Nearly a year later a saddle of bone covered the loins, and there was a series of lumps, apparently bony, on the margin of each latissimus dorsi, each teres major, the long head of the right triceps, in the left trapezius below the occiput, in the middle of the right trapezius, and in the supraspinous fossa. The boy also had a curious malformation of the head of each first metatarsal bone, which was large and prominent, while each great toe was small, consist-

ing, apparently, of but one phalanx, and directed toward the fibular side of the foot.

The nature of myositis ossificans is not well understood, hereditary predisposition being the most satisfactory explanation of its cause. The subject has been quite fully discussed by Seidel, in Gerhardt's *Kinderkrankheiten*, Bd. v. Abth. 2; by Pinter, in his *Inaugural Dissertation*, Wurzburg, 1883; by Mays, in Virchow's *Archiv*, Bd. lxxiv., 1878; and by Rogers, in *The American Journal of the Medical Sciences*, vol. xiii., 1833. All of these papers are referred to by Mr. Sympson, who cites other writers on the same subject, but leaves the subject about as obscure as he finds it. The most unfortunate aspect of the whole discussion is the fact that so far no satisfactory method of treating this curious disease has been discovered.

ASPIRATION OF THE STOMACH.

IN the *Dublin Journal of Medical Science* for January, 1887, FOY reports a case of alcoholic coma in which he drew off from the stomach, with an aspirator, a quantity of claret-colored liquid. The man had taken claret and whiskey very freely, and for two hours every effort had been made in vain to arouse him. When seen, he was totally devoid of sensibility, the extremities were cold, the face livid, the pupils widely dilated, respiration hardly detectable, no pulse, and the heart-impulse scarcely to be felt. He was bled to sixteen ounces, and while the blood was flowing a fine trocar was thrust through the abdominal wall, upward, backward, and outward from its point of entrance at the sternal end of the eighth rib, and the contents of the stomach aspirated. The quantity of ingesta removed was not stated, but the organ was not emptied, lest the canula should slip and fluid escape from it into the peritoneum. The heart commenced to beat, and respiration was restored. The stomach was then washed out, and soon afterward the man was able to speak, and the next day was well enough to go home.

The operation is defended on the grounds that the respirations were too feeble to permit of the introduction of the stomach-tube, and that apomorphia would not act in a condition of alcoholic coma.

As stated by Dieulafoy, aspiration of the stomach was performed by Paul in 1873, in a case of opium-poisoning in a child. It has also been used in cases of extreme dilatation of the stomach from stricture of the pylorus. In the case here noted we are inclined to attribute the man's recovery to the bleeding rather than to the aspiration, and it is possible that by waiting for the effects of the bleeding, the stomach-tube might have been passed without danger, and the risk of an abdominal puncture avoided.

A NEW laboratory for the furtherance of medical investigation, which will be built in Brooklyn next spring, promises to be a model institution of its kind. Exteriorly the building will present an imposing appearance, while within the arrangements will have the advantage of a careful study of those of the Johns Hopkins University and elsewhere. Being the outgrowth of private munificence, this institution will be welcomed as another indication that medicine in this country has passed into the era when it will, more often than formerly, share in the endowment gifts of the public-spirited and wealthy.

The new institution will be known as the Hoagland Laboratory, named after its founder, Dr. C. N. Hoagland, of the above city, a physician by education, not in practice, but who has at heart the advancement of scientific medicine. The plan includes a lecture-hall, rooms that will accommodate medical gatherings, and the foundation of a library; and the situation will, doubtless, become the rallying point of a variety of professional interests.

FOR a quarter of a century VIRCHOW has occupied a prominent place in German politics and, as an ardent supporter of liberal principles and of constitutional government, he has naturally been in constant opposition to Bismarck. Though not an orator, he is a concise and forcible speaker, and his opinions always command the attention of the House. He has endeared himself to the electors of Berlin by an extraordinary devotion to their interests, and in the City Council he has been for years the moving spirit in various schemes of municipal reform. In the election at present going on, consequent upon dissolution of the Reichstag by Bismarck, one contest will be watched with peculiar interest, Von Moltke against Virchow, absolutism against liberalism—the victorious leader of the greatest of modern armies against one of the most remarkable philosophers which this century has produced. Let us hope that our great teacher will be successful.

A CONTAGIOUS disease hospital is greatly needed in Brooklyn. A bill has been prepared for the State Legislature, which is designed to authorize the construction, within the city, of a hospital building that will be equal to the demands of the time in that direction. The present smallpox hospital is an antiquated structure, unsuitable for any but the pauper class, even if suitable for that. It is beyond the city, in the town of Flatbush. It is under the control of the Charities' administration, not that of the health office. Its record as to the mortality of its inmates at times runs unduly high. This condition of affairs has been pointed out, year after year, by the health authorities, and is about the sole remaining deficiency in the hospital service of the commu-

nity, but the necessary endowment by the Act of the Legislature has never been secured, although repeatedly applied for.

In a large city there is always a large number of persons, living in hotels and boarding-houses, domestics and others, who, when sick of a contagious malady, should have isolation and hospital treatment; many of them are able and willing to pay for accommodations like that to which they have been accustomed.

THE *Journal d'Accouchements* of January 15th, contains the statistics of the Maternity at Liege for the year 1886. It appears that during the year there were 417 women delivered, not one of whom died, and there were only three who suffered from septic infection, the type in all three being mild. Yet there were several operations done, for example, sixteen applications of the forceps, five internal podalic versions, and in nine women premature labor was induced; there were five cases of placenta prævia, and there were seven women with albuminuria, one of whom had eclampsia. Nevertheless, in 87 per cent. the puerperium was in all respects normal.

Such remarkably favorable results the director, DR. CHARLES, attributes to the faithful use of corrosive sublimate, which he prefers to any other antiseptic. That in only 13 per cent. of 417 women delivered in a maternity there should be a temperature of more than $100\frac{2}{5}°$, is certainly extraordinary, and seems at once a strong vindication of antiseptic obstetrics, and an example for other maternities, in which like results may be obtained by the faithful use of like means. The use of corrosive sublimate was begun in the Liege Maternity in May, 1884, and up to January, 1887, there have been 987 women delivered without a single death from septicæmia.

THE late Dr. Dudley, of Brooklyn, left a bequest of $500 to the Faculty of the Long Island College Hospital, to be used in encouraging their students to diligence in special study. The interest of this sum will each year be given to the senior year student who shall present the best thesis based upon some case that has been clinically observed during the year.

SOCIETY PROCEEDINGS.

NEW YORK SURGICAL SOCIETY.

Stated Meeting, January 12, 1887.

THE PRESIDENT, CHARLES MCBURNEY, M.D., IN THE CHAIR.

DR. J. D. RUSHMORE, of Brooklyn, read the following paper on

THE TREATMENT OF FRACTURED PATELLA.

The lack of certainty in the results of treatment in cases of fracture of the patella, and the different values that are put by writers on the subject upon the various methods of securing the fulfilment of the general indications in the management of this injury, with a desire to learn what experience has taught the members of this Society in the matter, are the reasons for submitting to the Society this evening for discussion a short paper on the treatment of fracture of the patella, in order that the conclusions drawn from cases that have been under my care may be either confirmed or corrected.

That the discussion may be as practical as possible, and, at the same time, confined within reasonable limits, I submit the question, What is the best treatment in recent cases of simple transverse fracture of the patella? An answer to this question would, doubtless, be, in the main, the same by each of us; yet we would differ as to the relative importance of the obstacles to be overcome, and the methods available for the purpose.

It is important to remember, in the first place, that the patella lies loosely in the tendon, when the limb is lying in a horizontal position with the muscle relaxed; that the bone can be moved by passive motion freely and easily in a lateral and a downward direction; and, not only so, but can be moved upward, as well, a distance of at least a third of an inch, showing that the ligamentum patellæ is not on the stretch normally; and so free is this motion of the bone, that the leg can be flexed on the thigh to an angle posteriorly of about 130 degrees before the patella becomes fixed and invulnerable. And I have never been able to satisfy myself that this motion was any freer in my own limb, with the body and thigh approximated as they necessarily are in the sitting position, than in the limb of another person, lying on a bed in a horizontal position, although theoretically it ought to be so. The quadriceps muscle also has insertion in the capsular ligament, as well as into the tubercle of the tibia, through the ligamentum patellæ. A considerable part of the vasti muscles converge, it is true, to be inserted into the patella; but there is, after all, a certain part of the tendinous tissue directly continuous with the thin, but strong, capsular ligament. A very little dissection will demonstrate this to the eye; and the power to render tense the ligament is shown by traction on the vasti muscles, in the dead subject; and a still better evidence of the force exerted by the vasti muscles in extension of the leg, in the living subject, is manifested by placing a finger on each side of the ligamentum patellæ, and making the effort necessary in extending the leg, when the ligament will be found not only to become tense, but will perceptibly broaden under this effort. This free motion of the patella is as marked, however, when the limb is horizontal as when it is elevated with the idea of approximating the origin and insertion of the muscle. It seems to me that these points are of value in deciding on what measures we shall use in diminishing the amount of separation that exists when the bone is fractured.

The production of a few cases of fracture of the patella in the dead subject, has thrown some light on the conditions essential for separation to take place in the fragments. I have been struck, in the first place, by the difficulty of producing a fracture of this bone by direct violence even with the leg flexed; and when the bone is sawn partly through from within outward, it is still difficult by any force applied on its external surface, to complete the solution of continuity. When the par-

tial section is made in the opposite direction, from without inward, a slighter force will complete it. When the bone alone is fractured, without any injury to the ligamentous and aponeurotic tissues adjacent, the amount of separation of the fragments is practically nothing; the moderately thin handle of an ordinary scalpel in a post-mortem case can, with difficulty, be inserted flatwise between the fragments. This amount of separation, as we all know, can be increased by cutting the soft tissues at the side of the bone; but even when the capsule was cut to the extent of three inches, laterally, on each side from the edge of the patella, the fragments could not be separated more than three inches and a half, and this required some pressure to be made on the lower fragment; additional separation could be obtained only by further lateral cuts and by vertical incisions upward.

The few attempts that I have made to produce separation of the fragments of fractured patellæ, by injections into the joint, have been almost entirely failures, whether employing air or fluids; for, in order to allow of the separation taking place at all, the aponeurotic tissues of the joint must necessarily be cut more or less, and, although the nozzle of the syringe can be tied in securely, the air or fluid injected, with even slight pressure, finds its way into the areolar tissue outside of the joint cavity, and distends its meshes very rapidly. It seemed to me, however, that there was a slight separation produced by the fluids that remained in the joint, but of this I cannot be sure. Of course, when the bone is broken in the living subject and inflammatory changes obliterate, in part at least, the areolar spaces in the immediate neighborhood of the lacerated capsule, and the fluid accumulates slowly, the problem is somewhat different, and under these circumstances the fluid might act in a way that it would be impossible for it to do in the dead body. I have an impression also, but nothing more, that when the limb was elevated the upper fragment sank a little downward by the weight of the water that remained in the upper part of the synovial sac; I should be unwilling, therefore, to draw any conclusions from my attempts to separate the fragments of broken patellæ by intra-articular injections.

I desire also, in connection with this subject, to describe the post-mortem appearance in two cases of fractured patella, in the Brooklyn Hospital, where death resulted from other causes but within a few days after the production of the fracture. These appearances are, of course, familiar to all of us, but an ocular demonstration of them adds interest to the matter under discussion, and these are the only opportunities that I have had to examine such cases. I can save time and space by speaking of them together.

They were middle-aged men; the fracture was in the right patella; recent in each (within three or four days of death); one known to have been produced by muscular action, and the other probably so, for the patient was intoxicated at the time of injury, and knew nothing of the method of production, but there were no marks of bruising in the soft parts over the joint, except a discoloration near the biceps tendon, the skin was somewhat less movable over the fractured, than over the sound patella; there was a separation of the fragments, of about one and a half inches; the circumference of the knee was one inch in the first, and one inch and a quarter in the second case greater than in the other knee; the lower fragment sagged down to a very slight extent; the fracture was a little below the middle of the bone, and transverse, and the edges felt sharp and were clearly defined.

On cutting into the joint a small subcutaneous clot was found in the first case, and a clot as large and as thick as the palm of the hand, in the second case, over the biceps tendon; the areolar tissue over the fragment was stained with blood, and to some extent matted to the tendinous covering of the upper fragment; the joint had been directly opened by a lacerated wound on each side of the patella in each case about two or two and a half inches in length, and in addition, in the second case, there was a vertical rent, partly through the vastus externus and through the synovial membrane, about three inches in length; there was a moderate amount of bloody serum in each joint, and, in the second case, a few small clots; the upper fragment in the second case was more movable upward than in the first case. Much to my surprise, the fractured edges were not sharp and clean-cut as I had supposed from my examination before the joint was opened, but the rent in the periosteum in front was at a lower level than in the bone itself and quite irregularly lacerated, so that it dropped down like an apron in front of the free edge of the upper fragment, partially covering the broken surface. This was true of both cases, and in the first case there were a few particles of bone adherent to the under side of this periosteal flap. The edges of the fracture were covered and concealed by a firm clot that required considerable pressure with the thumb-nail to remove it, and this, with the periosteal flap already spoken of, was undoubtedly the explanation of my inability to obtain crepitus, although the fragments could be brought into contact with each other. I take this peculiarity in the fracture to be unusual, not finding it described; but it is none the less singular that it should have been found in the only cases that I have had an opportunity to examine after death.

To consider briefly the condition that obtains in the living subject, we may start with the statement that the amount of separation of the fragments depends on the amount of laceration of the ligamentous and tendinous tissues in the neighborhood of the patella, not that in every case of extensive laceration there must necessarily be a wide gap, but that in every case of wide separation there must be extensive laceration as an essential condition. The presence of a considerable separation on superficial examination is proof of a considerable laceration, and we can satisfy ourselves of the presence or absence of laceration in cases where the fragments are close together by gentle flexion of the leg in the thigh, when the quadriceps will be excited to contract, and tend to draw up the upper fragment, while the lower fragment will be displaced downward in cases where there is any laceration. If there is little or no separation under these circumstances, we may know that there is little or no laceration. I have occasionally, with the same object in view, made gentle pressure upward and downward on the fragments, but it has been accompanied by rather more pain than by flexion.

If we now come to consider the causes of separation of the fragments, while we admit that the clot interferes temporarily with the close approximation of the frag-

ments, and the periosteal flap may, in some cases, prove a permanent obstacle to bony union, we must recognize that the real causes of separation are either muscular action or fluid accumulation into the joint, and it seems to me that facts do not warrant us in excluding either one of these causes. It does not seem to me that there is any natural tendency for the muscle to draw the upper fragment upward, inasmuch as the bone is so movable in its tendon; but when fracture takes place, the quadriceps is not excepted in the contraction that takes place in all voluntary muscles after fracture, produced probably either by direct or reflex irritation, contraction that, unless overcome by proper methods, causes a very considerable amount of permanent shortening. There are also frequent contractions of the muscle whenever the patient attempts to sit up, or even to turn, or to raise the hip, etc., for while the muscle is comparatively inactive after fracture, I do not think that it is paralyzed, for with slight stimulus the upper fragment is perceptibly drawn upward by it. Free from any tendency, therefore, to draw the unbroken bone upward, it seems to me when fractures have taken place, that spasmodic action and the usual contraction that occurs after any fracture are causes at work in the production of the gap in fractured patella. And these two kinds of action take place quite as readily with an elevated as with a horizontal limb. It is true, also, that in the few cases of separation of the ligamentum patellæ that I have seen, the bone has been drawn upward, producing a perceptible depression at the point of rupture, even though the joint was not injured, and the amount of fluid in the synovial sac did not seem adequate to account for the displacement.

That the fluid in the joint, however, is a potent element in the production of separation is to my mind quite clear. I saw it very forcibly illustrated in a case of fracture in the Brooklyn Hospital, while interne in that institution. An officer, in helping to transfer an injured patient from a carriage to the hospital entrance, slipped and fell, producing a fracture of the patella. I saw him within five minutes of the receipt of the injury, and found no separation. The fluid accumulated in the joint, and the separation increased as the fluid increased. One would expect that if there were tension of the muscle present, the separation would have taken place immediately; and it might be said that the usual behavior of muscles after fracture would account for the gap as well as the fluid in the joint; but as the fluid was absorbed, the gap diminished, as is not infrequent, and this, I think, could never have been due only to the diminished pressure on the fragments by the fluid in the joint.

Important, therefore, as the fluid in the sac is as a cause of separation of the fragments in the fractured patella, it seems to me a less potent cause than the unusual action of the muscle. For what I saw in a case a few weeks since, is generally true in cases that I have observed: that while the fluid had increased the circumference of the knee by two inches beyond the normal measurement, and aspiration had been suggested, by careful and slow traction on the fragments crepitus could be obtained, although the fragments were quite two inches apart. It must be remembered that the normal capacity of the joint is increased by the rent in the soft tissues, and the opening up to some extent of the areolar spaces in the neighborhood. Of course, it goes without saying, that the inflammatory trouble in the joint enters as a somewhat important element in the treatment of the fracture.

The indications for treatment are plain enough with regard to the coaptation of the fragments; the other indications usually mentioned are the treatment of the inflammation of the joint and its resulting effusion, and the prevention of ankylosis. The first is by all means the most important, and the variety of devices used for the purpose shows, in the main, their inefficiency. It seems to me, in the first place, that elevation of the limb is unnecessary and undesirable for the reasons already given. All those methods for approximating the fragments that make traction on the skin alone, and indirectly only on the bone, are very inefficient in bringing the edges of the fragments together. They have the advantage of early application, of not pressing downward, or tilting the fragments, and thus avoiding the formation of adhesions; but being attached only to the movable skin, they must fulfil very imperfectly this first indication.

Those appliances that are used in which traction is made obliquely backward, and downward, and upward on the fragments, while more powerful and efficient than the former method, have some objections—they cannot be applied until the inflammatory trouble subsides and the fluid is in part at least absorbed, or, if applied earlier, they produce so much pain that the necessary force cannot be used to approximate the fragments, and while pressing on the fragments they at the same time press on the fluid, and in both ways tilt the fragments up; and even when applied late, they press the fragments against the condyles of the femur, and favor the development of adhesions; nor have they a very good control over the upper fragment, for the least involuntary effort at contraction obliterates the depression above the patella, and the bone slides up underneath the dressings. I think the objection made to them by Marinegs has force also, that the constriction of the dressings presses on the nutrient arteries and so interferes with repair.

The most efficient way of approximating the fragments and keeping them in contact, is by traction directly on the bone, and it seems to me that Malgaigne's hooks accomplish this end satisfactorily. They can be applied very early, the traction is made directly on the bone fragments, and is in the long axis of the limb, thus avoiding both tilting and adhesions to the condyles of the femur, the fluid in the joint assisting in lifting the fragments up away from the condyles, the control of the motion in the quadriceps is complete; the fluid does not interfere with their application; they therefore give the best chance for bony or very short ligamentous union. The objections, it seems to me, are not at all sufficient to deter us from using them, in view of the advantages they possess. I have used them in five cases with success, getting, as an immediate result, a shorter bond of union than by any other method, though never union by bone thus far. The pain was not great, the location of the introduction of the points not being specially a sensitive part, and the wound behaving kindly by constant care and cleanliness. The pain of introduction could be diminished by the use of cocaine subcutaneously. The danger of necrosis, of

erysipelas, of suppuration, etc., is undoubtedly to be taken into account, but must be of very rare occurrence.

The objection that, if their use is not followed by bony union, the patient is worse off than if he had a moderately short ligamentous union, on account of the greater liability of fracture, seems to me not to be a fair one, for the cases of refracture or rupture under these circumstances have occurred soon after the discharge of the patient, while the bond of union was still weak; and we have all seen the same thing take place in cases where the separation was a half inch or more in length, the firmness of the ligamentous band being dependent rather on its age than on its length. The objection that they are "infernal and barbarous" is not sufficiently exact to be answered.

The second indication—the treatment of inflammation and the fluid accumulation—has been fulfilled in may cases by the ordinary rest, evaporating lotions, etc. I never have found it necessary to aspirate the joint, and it must be very seldom necessary to resort to this measure, as the broken fragments can be quite easily approximated with the joint quite distended with fluid; besides, the operation is not entirely free from the dangers of suppuration in the knee-joint.

It seems to me unwise to resort to any active measures in fulfilling the third indication—prevention of ankylosis by flexion ever so gentle, before the eighth week at the earliest, and to do this by the third week as is recommended, is dangerous to the integrity of the ligamentous union, if sufficient force is used to affect at all the adhesions.

I should expect, and have obtained the best results in the treatment of single transverse fracture of the patella by applying a posterior splint with the limb horizontal, and lying in a natural position, but not fully extended; by using Malgaigne's hooks, introduced on the first or second day after the receipt of the fracture; by keeping the dressing on for four weeks, then removing the hooks but not the splint, which has been kept on for four weeks more, the patient being in bed; then the patient allowed to be out of bed with the knee immovable for four weeks longer; by the use of the ordinary remedies for the relief of the inflammatory joint-symptoms in the early stage, but without resort to aspiration; and by avoiding any effort to disturb adhesions until after the end of the third month, and then only by the patient's ordinary use of the joint in walking, etc.

I have purposely omitted saying anything about unusual forms of fracture of the patella, because they are outside the scope of the present inquiry; and have also said nothing of the treatment of old cases with weak knee by wiring because I have no personal experience to relate.

DR. ABBE exhibited a specimen and related the case of a young lady who suffered an oblique fracture of the lower third of the patella, the fragments being widely separated. As she was very anxious to have a sound limb, and was willing to take the risk, he exposed the bone, under strict antiseptic precautions, and found a fracture, with two inches separation, the fractured surface of each fragment being covered by a dense layer of torn white fibrous tissue. This was the normal prepatellar tissue torn like two fringes, and so disposed over the bone that osseous union would have been impossible, even if coaptation had been obtained. These fringes were cut away. The patella was wired with silver, the joint irrigated with sublimate, 1 : 5000, and drained laterally. Primary union, without joint disturbance, followed. Three months have elapsed, and the patella is one solid bone. The patient is walking with a cane, and without splints. Fibrous ankylosis exists, which bids fair to yield in the course of time, and seems largely extracapsular, but at present allows flexion of about only ten degrees. The patient is greatly pleased.

He thought there was always some risk of ankylosis following this method, but that with improved technique this would be done away with. One step toward improvement would be in leaving a drainage tube in the joint interior as short a time as possible, and perhaps do away with it altogether. In the worst event he believed a partly ankylosed joint with solid patella, better than a weak unreliable knee with gaping fragments. If a joint suppurated, he believed it was always due to contamination from defective antisepsis.

DR. LANGE said that he had heard, with much interest, the reference to the fibrous tissue that was found covering the ends of the fragments, since he had noticed the same appearance. He had performed the operation of wiring the patella in five cases, and believed that it is safe and reliable, provided the surgeon exercise the proper care. He had so far operated on special indications only. The first patient was a stewardess on board a steamer, who wished particularly to have a reliable limb in order to be able to go up and down the steep ship-stairs, as her living depended on this. The second patient was a lady, who, for many years before, had had an amputation of the thigh on the opposite side. The third one had fractured both patellæ several years before; one was not operated upon. There existed a very wide gap, but the limb was partially useful. On the side of the operation the fracture had been of more recent occurrence, and though the distance between the fragments was considerable, the latter could, with some difficulty, be approximated.

In all of these cases, two of which were operated upon within eight or ten days after the accident, complete recovery took place, and the limbs have become quite useful.

The fourth case was that of a vigorous man, who, after a fracture near the attachment of the ligamentum patellæ, had lost all control over the extension of his leg, and was obliged to depend upon an apparatus. There existed a wide diastasis of the fragments, which could be brought in contact only after the quadriceps tendon had been partially divided at several points, at some distance higher up, between the muscular bellies of the vastus. The capsule was also incised freely. The fragments were forcibly approximated by coarse silk ligatures. This man had a stiff, but useful, limb. It was impossible to say whether the slight motion that was present at first, had improved or not. The shape of the patella and its lower surface had to be smoothed. Probably a comminuted fracture originally occurred.

The fifth case was not equally favorable. A strong man, aged about thirty, fond of athletics, had fractured his patella near the upper margin. There existed extensive extravasation and rupture of the capsule. The operation was done on the third day after the injury,

the patient himself being anxious to secure the best possible result. Borosalicylic solution was used as an antiseptic during the operation. The patient did not behave as quietly as he might after the operation, and made very unwise movements with his limb. Suppuration set in, and the limb became quite stiff, the semilunar cartilages having been detached by necrosis. Probably it would have been wiser to delay the operation until absorption of the extensive extravasation had taken place. In all of the cases, except one, strong silkworm gut was used to suture the fragments.

DR. HALL stated that he had wired the fragments of the patella in two cases of recent fracture. In both instances he had found coagulated blood between the edges, the extravasation in one case being very extensive and having ruptured into one of the prepatellar bursæ, so that the patella could with difficulty be felt. Aspiration was unsuccessful as the blood was coagulated, and fearing much stiffness on account of the hæmarthrosis, suturing was resorted to. Bony union was obtained in both instances. Silver wire of one-sixteenth of an inch was used. The special interest of the cases lay in the fact that there was no interposition of fibrous tissue, the fragments being separated to the extent of half an inch by a firm blood-clot; particular attention was called to this, as the other condition had been described as invariable, and as being the cause of non-union. In a case of old fracture recently seen, only the upper corner of the bone was broken off, with practically no separation, and no interference with the insertion of the quadratus femoris into the larger fragment, so that the patient had perfect use of the leg, although union did not occur.

DR. SANDS asked concerning the amount of motion in the injured limbs.

DR. HALL replied that one of the patients could flex the limb only about fifteen degrees when the dressings were removed, but three or four weeks later, when last seen, had motion almost to a right angle, and was improving daily. No passive motion had been employed. In the other case flexion was limited to about twelve degrees, but the range of motion would undoubtedly be greater in the course of three or four months.

DR. PETERS asked how long the dressings were retained, to which the speaker replied, for six or seven weeks.

DR. LANGE remarked that he had obtained good results with sutures of coarse silkworm gut.

DR. SANDS thought that all surgeons had found the treatment of fractured patella unsatisfactory, it being impossible to maintain perfect apposition of the fragments. Treves had recently recommended anew the use of Malgaigne's hooks, piercing the joint and embedding them firmly in the fragments, due antiseptic precautions being employed. The speaker rather liked this treatment. After all, it is not so important to obtain bony union, as the patients can get on very well without it. However, the best results will doubtless be obtained when surgeons are able to open the joint safely every time, and to wire the fragments. It should be remembered that this is yet a serious operation, which is sometimes followed by suppuration, ankylosis, or even by death. He had found that patients rarely wished to run any risk, when the nature of the operation was frankly related to them. Even when successful the operation often leaves some stiffness of the joint, with diminished flexion, on account of the thickening which attends the healing process. Dr. Sands confessed that he, at present, occupied a decidedly conservative position with regard to the operation and would be unwilling to have it performed upon himself. But he was confident that the dangers now attending it will be finally overcome.

DR. WEIR explained that Mr. Treves's method of introducing the hooks is quite simple. He relied upon the maintenance of perfect cleanliness by means of water.

DR. HALL believed that a Philadelphia surgeon had written a paper some years before, in which he reported about fifty cases of fractured patella, in the treatment of which Malgaigne's hooks had been used with good results.

THE PRESIDENT thought that the radical operation should be more carefully studied before it is generally advised, since its remote consequences are not always satisfactory. He had observed a case in which bony union had been obtained by wiring the fragments, but three months after they separated, on the application of slight force; in another instance suppuration occurred, and motion was subsequently limited to fifteen degrees. Another case of suppuration was reported at Bellevue Hospital.

DR. POORE asked if it was not true that the joint is frequently stiff after the operation.

DR. LANGE replied that this depended to a great extent upon the after-treatment. In three of his cases there was perfect mobility, while in the others motion was limited. The condition doubtless improves in time under the influence of massage and passive motion.

CORRESPONDENCE.

CHLOROFORM AND ETHER DEATHS.

AN article published by me in THE MEDICAL NEWS for January 8 was intended simply to put upon record a clinical experience, which seemed to me important as demonstrating that danger attended the administration of an anæsthetic mixture generally regarded as safe. Among the remarks appended to my account of that case was one calling in question the statement made by Dr. J. C. Reeve, that ether "in the human subject may cause death as suddenly, as unexpectedly, and in the identical manner that chloroform does." Dr. Reeve, in an article in THE NEWS for January 22, asserts the accuracy of his statement, and adduces a number of instances in support of it.

I beg to disclaim any intentional discourtesy to Dr. Reeve in my expression of dissent from his opinion; nor did I mean to throw down a challenge upon the subject. But I feel constrained to defend my own view, that there is a marked difference between ether deaths and chloroform deaths; and shall call Dr. Reeve's own cases in evidence.

Let me first very briefly review these:

In Case I. there was "an extremely softened spleen."

Case II., a woman, "aged fifty-five, in very poor health."

As to Case IV., Dr. Reeve says "the report of the autopsy makes no mention of" blood in the trachea. Certainly not, for in the account (in the *Boston Med.*

and Surg. Journal for November, 1875, taken from the *N. Y. Herald*) there is no mention of any autopsy.

In Case V. there were "Bright's disease, chronic pleuritis, and embolism of the pulmonary artery."

Case VI. (Lowe's case). As to this, I must speak a little more fully. Dr. Reeve says of it: "The report I have says 'there were some nodules of cancer in the liver and lungs.'" My own quotation was made from Mr. Lowe's original statement in the *British Medical Journal*—"studded throughout both lungs, but more particularly toward the base, were numerous small, hard, cancerous nodules."

Case VII. was that of a "male, aged sixty-nine, much exhausted; strangulated hernia; heart flabby, left ventricle not contracted, lungs emphysematous, and bronchi filled with purulent mucus."

In Case VIII., that of a man, aged sixty-two, addicted to drink, there were found "pleuritic adhesions, atheromatous aortic valves, congested liver and kidneys, the latter studded with cysts."

Case IX. was that of "a feeble old man of sixty-seven, with strangulated hernia." It seems to me that this was a case of "asphyxia," as stated, by the "copious bronchial secretion" which "kept filling the patient's mouth." Such drowning is sometimes met with in various thoracic affections of old people.

In Case XI., that of a man, aged sixty-two, "the lungs were emphysematous and congested, and there was bronchitis (?). The heart was flaccid and fatty, with adherent pericardium."

In Case XIII., that of a man, aged fifty, "suffering from severe inflammation of the bowels, due to obstruction," there was found at the post-mortem "considerable purulent effusion in the peritoneal cavity; at the lower part of the descending colon was a malignant mass of the size of a small cocoanut, blocking the canal. In the right ventricle of the heart was a fibrinous clot attached to the tricuspid valve."

The other cases are all deficient, as indeed Case IX. was also, in statement of post-mortem appearances. But in Case XII., the only one in which a young person was the victim, it is stated that the girl, aged ten, was "much emaciated, and looking very ill; to be sounded for a calculus."

Now, I would ask to contrast with this series of cases a few only of those of chloroform death which have met my eye in the hasty glance over some medical journals which the pressure of other work has allowed me to take.

I. A stout young man operated on by Dunsmure for stricture of the urethra. With the exception of a few old pleuritic adhesions, every organ presented the most exquisite specimen of health. (*Med. Times and Gazette*, October 15, 1853.)

II. Young woman, chloroformed for an amputation of the great toe. All the organs found perfectly healthy. (*New Orleans Med. and Surg. Journal*, January, 1854.)

III. Richard's case of uterine polypus. Nothing of interest observed at the post-mortem. (*Lancet*, April 29, 1854.)

IV. Man, aged forty; eyeball excised at the Royal Ophthalmic Hospital, London. No cause of death discovered at the autopsy. (*Med. Times and Gazette*, April 14, 1855.)

V. Porter and Brewer's case; trooper, with dislocation of thumb. Nothing detected at the post-mortem except "congestion of the scalp and head." (*Maryland and Virginia Med. Journal*, February, 1861.)

VI. Lane's case; boy, aged eight, to be operated on for the scar of a burn. All the organs found healthy. (*Med. Times and Gazette*, November 16, 1861.)

VII. Healthy looking man, aged thirty-three, with a sloughing sore on the penis to be cauterized. No sign of disease discovered at the autopsy. (*Lancet*, November 19, 1864.)

VIII. My own case. Man, chloroformed for the second time for reamputation of leg. No visceral lesions sufficient to account for death. Heart and lungs perfectly healthy. (*Am. Journal of the Med. Sciences*, January, 1865.)

IX. Woman, who had previously taken chloroform; had to have a tooth drawn. At the autopsy all the organs were found perfectly healthy. (*Med. News and Library*, June, 1866.)

X. Le Fort's case. Man, aged thirty, with anal fissure. Nothing noted at the post-mortem but two small pulmonary cavities. (*Med. News and Library*, June, 1873.)

XI. Brakeman on a railroad, chloroformed for the extraction of a tooth. No disease found. (*Boston Med. and Surg. Journal*, October 1, 1874.)

XII. Gant's case. Boy, aged fourteen, with suspected dislocation of the hip. All the organs sound. (*Brit. Med. Journal*, December 19, 1874.)

XIII. Man, aged fifty-six, operated on for fistula and hemorrhoids. No organic disease discoverable. (*Brit. Med. Journal*, March 17, 1877.)

XIV. A young lady who had had five teeth extracted under chloroform, was recovering, when more chloroform was given, and she died. A post-mortem examination showed all the organs to be healthy. (*Med. News and Library*, March, 1878.)

The above cases are only selected as having distinct statements in regard to the post-mortem appearances; otherwise they are taken at random. By reason of their incompleteness in this respect, I have not noted here such cases as one mentioned by Anderson (*Cincinnati Clinic*, March 31, 1877), in which a strong, healthy country boy, who had taken chloroform a year previously, inhaled it (for some purpose not stated) and was recovering, when a few more whiffs were given him with fatal effect; nor another recorded in the *Association Journal* for May 26, 1854, where a girl of thirteen, of strong constitution, was chloroformed for the removal of a lipoma of the back, fell forward, and died in a few minutes. Nor have I regarded any cases of self-administration. Yet, so far as my knowledge goes, all these cases, like those above adduced by me, are without parallel in the history of anæsthesia by sulphuric ether.

I am not aware that I have ever denied the occurrence of deaths during the administration of ether, or as a result of it; although young men do sometimes make assertions which more mature experience leads them to correct or modify. But with nearly all the cases brought forward by Dr. Reeve I had been familiar, as well as with some others not mentioned by him; notably that of Dr. Dandridge, of Cincinnati, reported in 1880, and that of Dr. Roberts, of Philadelphia, of about the same date. And in every one of these cases, so far as I know them, in which an autopsy was made, there was

discovered serious organic disease, perhaps not in itself lethal, but too grave to be overlooked as contributory to the fatal result. Chloroform needs no such assistance.

Upon the production of one case in which ether has proved fatal to a healthy young person, inhaling it for a slight operation, and in which an autopsy shows no organic disease or lesion, I will at once admit that my objection to Dr. Reeve's statement falls to the ground, and that ether does kill in the identical manner that chloroform does.

In conclusion, I would quote a passage which by the merest accident caught my eye a day or two since. It occurs in a review of Valette's "Clinique Chirurgicale de l'Hôtel-Dieu de Lyon," in the *British and Foreign Medico-Chirurgical Review* for January, 1876:

"One lecture is devoted to a comparison of the relative safety of ether and chloroform, and M. Valette pronounces unhesitatingly in favor of the former. He does not deny that deaths occasionally occur under the influence of ether; but in the cases he has been able to collect, there is a remarkable difference between these and the similar accidents from chloroform. A large number of the fatal cases under chloroform have occurred when the anæsthetic was being administered to a patient apparently in good health, and suffering from some trivial local malady. On the other hand, those occurring under ether have always been in cases of the gravest character, and in which the patient was either exhausted by previous disease, or suffering from the shock of some severe injury. Again, he points out that death, when resulting from the effects of ether, has always been preceded by warning symptoms; whereas with chloroform it has often been sudden, and utterly without warning of any kind."

JOHN H. PACKARD, M.D.,
Surgeon to the Pennsylvania Hospital.

PHILADELPHIA, January 31, 1887.

NEWS ITEMS.

DISEASE AMONG THE INDIANS.—DR. WASHINGTON MATTHEWS, surgeon in the United States Army, has made a valuable contribution on the causes which are at work in carrying off the Indians of our country. One of the most important of these he finds to be consumption. From the census of 1880 we learn that, while the death-rate among Europeans is 17.74 per thousand, and that among Africans 17.28, the rate among the Indians is no less than 23.6. In diarrhœal diseases the Indian death-rate is not greatly in excess of that of the other classes. Measles gives a mortality of 61.78 per thousand. But it is under the head of consumption that the difference between the Indians and the blacks is most conspicuous; the rate among the former being 286 as compared with 186 among the latter, while among the whites it is but 166 in the thousand. Dr. Matthews finds that, where the Indians have been longest under civilizing influences, the consumption rate is the highest; meaning by the term "consumption-rate" the number of deaths from consumption in a thousand deaths from all known causes. Thus the rate among reservation Indians in Nevada is 45; in Dakota, 200; in Michigan, 333; and in New York, 625.

A PASTEUR LABORATORY AT NAPLES.—The Sanitary Commission at Naples has determined, upon the recommendation of Semmola, to erect a laboratory for the practice of Pasteur's method of inoculation. The circular which announces its erection, urges the need of further experimentation with this method, to enable an accurate estimate to be made of its worth.

INSANE DEPARTMENT OF THE PHILADELPHIA HOSPITAL.—Dr. A. J. Smith has been elected first assistant in the insane department of the Philadelphia Hospital, to succeed Dr. J. C. DaCosta, who has been called to a similar position in the Pennsylvania Hospital for the Insane.

FEMALE MEDICAL STUDENTS IN FRANCE.—The medical faculty of France has this year 108 female students—namely, 83 Russian, 11 English, 7 French, 3 American, 2 Austrian, 1 Turk, and 1 Roumanian.

DEATH FROM CHLOROFORM.—The death of a man, aged fifty-four, while under the influence of chloroform, administered for the purpose of performing an operation on a long-standing disease, recently occurred at Bradford, England. The evidence given at the inquest showed that every precaution was taken, and that an examination of the heart before the administration revealed no evidence of disease. An ounce of brandy was administered three-quarters of an hour before the administration of the chloroform. Immediately on the completion of the operation the heart's action was found to have ceased. It was stated that the deceased had successfully undergone a similar operation twenty-five years before.—*British Medical Journal*, Jan. 15, 1887.

TOOTH POWDERS.—According to *The Lancet* precipitated chalk forms the best basis for a tooth powder, to the base of which may be added pulv. saponis and ol. eucalypt. a drachm of each; and, if there is no objection to the taste, half a drachm of carbolic acid.

NOTES AND QUERIES.

SANDS ON USE OF PASSIVE MOTION.

To the Editor of THE MEDICAL NEWS,

SIR: The article written by me "On the Use and the Abuse of Passive Motion," contains, as published in THE MEDICAL NEWS of January 22d, numerous errors, for which I am not responsible. In transmitting the proceedings for publication, the Society's Recorder sent my original manuscript to the *New York Medical Journal*, and forwarded to THE MEDICAL NEWS a type-written copy which was not submitted to me for correction, and contains a number of errors which did not exist in the original.

Please to do me the favor of publishing this letter in the next number of your journal, and oblige,

Yours very respectfully,
H. B. SANDS.

NEW YORK, January 23, 1887.

OFFICIAL LIST OF CHANGES IN THE STATIONS AND DUTIES OF OFFICERS SERVING IN THE MEDICAL DEPARTMENT, U. S. ARMY, FROM JANUARY 25 TO JANUARY 31, 1887.

MORRIS, EDWARD R., *First Lieutenant and Assistant Surgeon.*—Granted leave of absence for one month, to take effect about March 10, 1887, with permission to apply for an extension of twenty days.—*S. O. 6, Division of the Pacific*, January 19, 1887.

CLENDENIN, PAUL, *First Lieutenant and Assistant Surgeon.*—Ordered for duty as Post Surgeon at Camp Pena, Colorado, Texas.—*S. O. 14, Department of Texas*, January 26, 1887.